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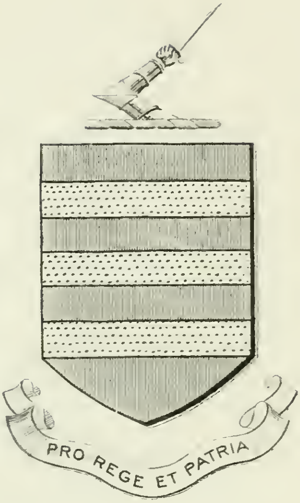

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THE JOURNAL OF NERVOUS AND MENTAL DISEASE,

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Original Articles, Selections and Translations.

Art. I.—ON THE STRUCTURE AND FUNCTION OF THE GANGLIONIC BODIES OF THE CEREBRO-SPINAL AXIS.

By Dr. H. D. Schmidt,
Pathologist of the Charity Hospital, New Orleans, and Member of the American Neurological Association.

In my paper on the "Structure of the Double-Bordered Nerve Fibre," published in the *Monthly Microscopical Journal* of London, May, 1874, I briefly alluded to the structure of the ganglionic bodies of the cerebro spinal axis, showing that it is similar to the structure of the axis cylinders of nerve fibres. Shortly after, in a communication to the Royal Microscopical Society, published in the July number of the same journal, and embracing, in the form of a synopsis, the principal facts, which I had elicited from a series of microscopical researches upon the structure of some of the nervous tissues, I again made some limited remarks upon the structure of these bodies, postponing the details to some more convenient time.

Since that time I have had ample opportunity of studying the subject still closer, and am now prepared to present he results of these studies to the profession, and in a more perfect and definite form.

To form a correct idea of the structure of the different nervous tissues, we must bear in mind that the whole nervous apparatus consists, in its central as well as in its peripheral parts, of only three essentially nervous anatomical elements. These are fibrils, nuclei, and granules, and, moreover, a certain intermediate so-called "inter-cellular" substance, by means of which granules, lineally arranged in rows, are united to each other and transformed into fibrils. The only difference existing between the individual nervous tissues consists in the morphological arrangement and mutual relations of these elements. The nerve-medulla and tubular membrane of the dark-bordered nerve fibres, though they may serve some special purpose, must be regarded as non-essential to the liberation and propagation of nervous energy.

In the cerebro-spinal axis, these ultimate anatomical nervous elements are supported and held in their mutual positions by an exceedingly delicate fibrillous connective tissue, the neuroglia, characterized by remaining unaffected by acetic acid. The axis cylinders of the peripheral nerve fibres and their peripheral terminal organs, together with the various ganglionic bodies of the nerve centres, are composed of one or more of these anatomical essential nervous elements.

The discussion of the structure of the ganglionic bodies, or ganglion cells of the cerebro-spinal axis, requires a few preliminary remarks relating to that of the axis cylinders of the dark-bordered nerve fibres, which, as we know, are but the direct continuations of the subordinate branches of the processes of the ganglionic bodies. All axis cylinders, whether surrounded by covering layers, as in the dark-bordered nerve-fibres, or whether appearing uncovered, as they are met with in the sympathetic nerves, consist of a number of individual nervous fibrils which, united by a homogeneous inter-fibrillous substance, are surrounded by a delicate, tubular sheath. Both the sheath and the inter-fibrillous substance appear to be continuous with each other, and are very probably of the same nature as the intermediate substance, by the agency of which the original rows of granules were transformed into fibrils during the earlier stages of the development of the axis cylinders in the embryo. As regards the individual fibrils of the axis cylinders, I repeat my former assertions in stating that they are not *smooth* fibrils held together by a granulous inter-fibrillous substance, as they were first described by Max Schultze, and as they are, perhaps, still looked upon by the majority of histologists; but that, on the contrary, they present a knotted appearance, and are held together by a homogeneous inter-fibrillous substance. The knotted appearance of these fibrils, indicating their *original anatomical composition of elementary granules*, can be recognized on all axis cylinders, whether large or small, fresh or hardened by reagents. For further details concerning the structure of the axis cylinder, I must refer to my paper on the "Dark-Bordered Nerve Fibre," already mentioned above, and also to my researches upon the "Development of the Nervous Tissues in the Human Embryo," published in the JOURNAL OF NERVOUS AND MENTAL DISEASE, July, 1877.

In passing from the axis cylinders toward the centre we, of course, arrive at the processes of the ganglionic bodies, and, as the axis cylinders represent nothing else but the subordinate branches of the processes, it becomes obvious that the latter are composed of the former, and that both are very probably also identical in structure. And this is really the case as far as their structural elements are concerned. In the mutual relation of these elements, however, a slight difference is already observed, which, bearing upon the propagation of nervous stimuli, deserves our attention. It consists in the individual fibrils not being applied to each other as closely in the processes as in the axis cylinders. In the processes, therefore, the individuality and the granulous character of the fibrils, indicated by the longitudinal and transverse striæ or furrows, becomes more apparent with the increase of the process in thickness, and the nearer to its origin from the body of the ganglionic cell. The component granules of the individual fibrillæ also, are rendered more distinct and prominent by a decrease of the intermediate substance which binds them together. Finally, upon the ganglionic body itself, the individuality of the granules, of which the fibrils are originally composed, is most distinct, so that the whole ganglionic body may appear to the careless observer as a mass of granular protoplasm—such as it was generally considered not many years ago.

While, however, upon the ganglionic body, the granules are seen more distinctly than upon the processes, the longitudinal furrows or inter-fibrillar spaces have not proportionately increased in width; and it is owing to this circumstance that the individual fibrillæ cannot always be traced very distinctly while passing over the body from one process into the other. This is particularly the case on those large ganglionic bodies of the anterior horns, which, on account of their bulk, do not transmit sufficient light through their centres as required for the examination. Nevertheless, even in these large specimens, the individual fibrillæ can almost always be distinguished and traced, as they spread over a considerable portion of the body. The course of these fibrillæ is most advantageously studied in those large, spindle-shaped ganglionic cells, met with in the anterior horns from which only a few processes are seen to arise (Fig. 1). The individual fibrillæ of the processes and of the ganglionic body itself, as well as their granular composition, can be distinctly recognized under all conditions, whether the specimen be fresh and uncolored, or slightly hardened and colored. The best method, however, is that proposed by Gerlach, by which beautiful preparations may be obtained. In examining one of these ganglionic bodies of the anterior horns, prepared and isolated by Gerlach's method, as represented in Fig. 1, we should first direct our attention to the fibrillæ of one of the processes, and, after having them distinctly recognized and studied, follow them as they pass upon the surface of the ganglionic body. This can be easily accomplished in adapting, by a careful management of the fine adjustment of the microscope, the focus successively to every point of the fibrillæ, spreading themselves over the body of the ganglionic cell. In tracing then the fibrillæ of a neighboring process in the same manner over the latter, it will be found that they cross in more or less acute angles those of the first process.

In some instances the decussation of the fibrillæ of the two processes can be seen almost without altering the focus. If not, the slightest alteration of the latter will bring it into view. The fibrillæ of one process do not enter the body of the ganglionic cell in the form of a bundle, but diverge from each other, either in the form of a hollow cone, to be distributed while embracing the nucleus over the whole body, or radiating over a portion of the body only.

The distribution of the fibrillæ over the ganglionic body is most distinctly seen on those smaller, strictly spindle-shaped, ganglionic cells with only two processes, representing the opposite poles of the body. Here it will be observed that they diverge in the form of a hollow cone, around the nucleus; and, after having embraced this body, gradually approach each other again to form the opposite process. For a long time I have been in doubt whether the fibrillæ of the processes would be sufficiently large in number to surround the nucleus completely, or whether they were giving rise to additional branches to fill up the interspaces left. From numerous close examinations, however, I am now convinced that the nucleus is only embraced by the fibrillæ of the processes. In the smaller spindle-shaped ganglionic bodies, as mentioned before, the fibrillæ of one process may be traced, in many instances, directly into the other process. Not all the fibrillæ arriving at the ganglionic body through one process, pass over the ganglionic body to enter the processes arising on the opposite side; on the contrary, a smaller or larger portion may enter a neighboring process, and without ever having passed the nucleus, direct their course toward the periphery. This fact can be demonstrated without difficulty. In examining the fibrillæ directly around the nucleus, especially in the larger ganglionic bodies, they present the appearance of being concentrically arranged around this body, from which the conclusion might be drawn that the fibrillæ representing the axes of the processes encircle, to some extent, the nucleus, before they leave the ganglionic cell through one or the other process. As all fibrillæ which can be traced over the surface of these larger bodies, are probably derived from the periphery or surface of the processes, it is difficult to determine the exact course of those representing the axis of the processes. These may either, in preserving their individual fibrillar character, pass only over a portion of the nucleus, and then leave by way of another process; or it may be that in the vicinity of the nucleus, the mutual connection of their component granules becomes looser, the whole assuming the appearance of a granular mass. If this were the case, however, it would necessitate a difference in function between the axial and peripheral fibrillæ of a process. Therefore, it is more plausible to presume that the arrangement of the axial fibrillæ around the nucleus is the same as, or, at least, similar to that of those derived from the surface of the process. Several years ago I met with the remaining portion of a ganglionic body, which had been divided by the knife in the making of a section. As the section had passed close to the nucleus, several unbroken fibrillæ could be seen passing directly in front of this body, while fragments of others, resulting from the section, were observed in the vicinity. Fig. 3 represents the specimen. This observation alone would show that the fibrillæ simply pass over the nucleus without encircling it.

From the above demonstrations the conclusion may then be drawn, that the ganglionic body or cell is formed, in accordance to its size and form, by a smaller or larger number of nervous fibrillæ—mostly derived from the axis cylinders of the nerve fibres—embracing an independent body of a spherical or ovoid form, known as the nucleus. In embracing the nucleus, the fibrillæ of the different processes not only decussate, but probably also interlace with each other, particularly those in its immediate vicinity. While surrounding the nucleus, the fibrillæ form no direct communication with it, but remain isolated. The interspaces left between the fibrillæ of the ganglionic body are filled up by the same inter-fibrillous substance which connects the fibrillæ of the processes, or of the axis cylinders.

The nucleus of the ganglionic body differs from the *nuclei* of other tissues, in the complexity of its construction. It represents a vesicle filled up by a granular protoplasm containing in its centre a smaller vesicle, the so-called nucleolus. The vesicular nature of both the nucleus and nucleolus is indicated by the double contour which they present. The complexity of construction just mentioned, will be found in the nucleolus. This body, namely, wherever it is met with in the nuclei of other normal cells or tissues, is always homogeneous in its nature, representing a large granule, bordered by a single contour. In the nucleus of the ganglionic body, however, it appears in the form of a vesicle, distinguished by a fine and distinct double contour, and containing two minute bodies which are characterized by their reddish lustre; the remaining space is filled up by minute granules. The one of these minute bodies appears in the form of a vesicle of about $\frac{1}{600}$ mm. in diameter; it is also bordered by a double contour, and, containing a dark granule in its centre, is especially distinguished by its bright and reddish lustre. The other minute body is somewhat smaller, and sometimes absent. Very frequently several nucleoli are found in the nuclei of the larger ganglionic bodies; these, however, contain no vesicles, but are only filled up with minute granules. The form of the nucleus seems to be in accordance with the particular form of the ganglionic body, which, in its turn, is determined by the number of the processes, and by the direction in which they approach the nucleus. Thus, in those large, more or less round, ganglionic cells, the nucleus also presents a round form; while in others of an oblong shape, the nucleus assumes an oval or oblong form. In the larger ganglionic bodies of the cortical layer of the cerebrum, being somewhat pyramidal in shape, the nucleus, though mostly oval, nevertheless presents in many cases also a pyramidal form. It seems, therefore, that the form of the nucleus is to a certain extent determined by the course of the fibrillæ embracing it.

All ganglionic bodies of the cerebro-spinal axis are constructed in the manner described above. Even in the smaller kind, met with in the upper strata of the cortical layer of the brain, the individual fibrillæ of their processes may be distinguished. In many ganglionic bodies the course of the fibrillæ over the body is hidden by the accumulation of pigment granules, usually found near the origin of the largest process; but, even in these instances, it may be detected upon that portion of the body which is free from granules.

The larger ganglionic bodies are invested by a delicate sheath, extending over the processes, and being, moreover, continuous with the sheath of the axis cylinders. Upon the body of the ganglionic cell, the sheath is not easily demonstrated, for the reason, as I suppose, that it is closely united, or even fused with the inter-fibrillous substance: but upon the processes it can be seen: in many instances it manifests, upon the larger processes, its presence by a number of minute spindle-shaped nuclei, which it contains.

Before dismissing the ganglionic bodies of the cerebrospinal axis, I shall have to make a few remarks upon the probable destination of the subordinate branches arising from the division of the processes, to which I have already alluded, on another occasion, some years ago. Since the discovery of the so-called axis cylinder process by Deiters, the idea has been prevailing that this process, without undergoing any division, represented an axis cylinder from the beginning, which, not far from its origin, would be surrounded by a nerve medulla and a tubular membrane, and thus transformed into a dark-bordered fibre, was to leave the grey matter of the spinal marrow, and join the anterior roots of the spinal nerves. All nerve fibres, forming these roots of the spinal nerves, therefore, would receive their axis cylinders from this source. It is true, that from a number of the larger ganglionic bodies, one or two processes, with a diameter about equal to that of the axis cylinder of a large, double contour nerve fibre, are observed to arise; but whether the axis cylinders of all the fibres of the spinal nerves are derived from this source only, is by no means an established fact. As far as my own experience goes, I must confess, that in the numerous preparations which I have made and examined, I have never been so fortunate as to behold the transformation of this axis cylinder process into a double contour nerve fibre, as close to its origin as I have seen it represented on the paper. I always found the process torn. Nevertheless, I would not contest the possibility of such an occurrence. Whether the axis cylinders of all large nerve fibres are derived from this source, is a question which must be negatively answered, as I have, in several instances, observed large axis cylinders arising from the larger processes, and have traced them to a considerable distance. In one instance, I observed three axis cylinders from $\frac{1}{300}$ to $\frac{1}{200}$ mm. in diameter, arising from one of the larger processes, one of which attained a length of more than $\frac{1}{5}$ mm. before it was torn. As regards the smaller axis cylinders, I have observed some of $\frac{1}{400}$ mm. in attaining a length of nearly one millimetre: and others of only $\frac{1}{600}$ mm. diameter, attaining a length of from $\frac{1}{4}$ to $\frac{1}{2}$ mm. Those naked axis cylinders which are met with in every fragment of grey matter of the anterior horns, when prepared by a weak solution of chromic acid, or of its salts, and carefully dissected by means of fine needles, are undoubtedly identical with those arising from the divisions and subdivisions of the larger and smaller processes of the ganglionic body: and if in reality—with the exception of the so-called axis cylinder processes—none of them, after their transformation into a double contour nerve fibre, contributes to the formation of the anterior spinal roots, it is more than probable that the majority enter the anterior and lateral white columns. From the examinations which I have thus far made in regard to the particular direction of the various processes of the ganglionic bodies of the anterior horns, on thin, very transparent, transverse and longitudinal sections of spinal marrow, it seems that the individual processes do not point in a particular direction. Accordingly, I have seen the largest process, which cannot be easily mistaken, pointing in one instance, in the one, and in another instance, in the other direction. The same I have found with the axis cylinder process. As the axis cylinders arising from the processes do not pursue a straight course, but, on the contrary, describe greater or smaller curves, before leaving the grey matter, it becomes almost impossible to determine their destination by the direction in which their parent process points.

As regards the termination of the finest subdivisions of the processes in the grey matter itself, I have spared neither trouble nor pains to come to some definite conclusion. Some years ago, in my first communication to the American Neurological Association upon the structure of the nervous tissues, I stated in regard to this subject that I had never been able to discover the existence of a minute nervous net-work in the grey matter of the spinal marrow in which the finest subdivisions of the processes were supposed to terminate, and from which, moreover, nerve fibres were to arise, such as described by Gerlach. At the same time, I also stated that from a physiological point of view I had presumed the existence of this net-work; and as my examinations had been made on specimens hardened in a chromic acid solution and uncolored, I promised to repeat these examinations on specimens prepared by Gerlach's own method, at a future day. As besides Gerlach, other histologists have professed to have seen this net-work, and, as furthermore, its existence or non-existence has some important bearing upon the particular mode in which the spinal marrow operates, I shall not hesitate to give a minute account of the examinations which I have since made.

In the latter part of August, 1877, after having made the preliminary arrangements with a butcher in this city, I drove to his yard in the evening, near sundown, when he was ready to kill his calves, carrying with me not only a solution of bichromate of ammonia, strictly prepared after Gerlach's formula, but also a similar solution of bichromate of potassa. The calf was killed and the spinal marrow removed without injury by the butcher, with the help of his sharp hatching knife, as neatly as and more rapidly than I could do it myself. I quickly removed the pia mater from the cervical enlargement of the still warm cord, and with my section knife, almost sharper than a razor, made a number of longitudinal sections of the anterior horns, which were immediately put in the above-mentioned solutions. On my returning home soon after they were put in a cool place, where they remained for two days; after which time they were transferred to the solution of carmine. Twenty-four hours after this time I commenced my examinations, which, in order to observe the gradual changes which might be caused by the ammoniacal solution of carmine upon the tissues, were repeated during five days. These examinations throughout were made in the most exact and careful manner. I am accustomed to work with needles, the points of which, about four millimetres in length, are very slender and bent at an angle of 45°. The very point, which I finish myself upon a hone, is examined during this process with a strong loupe, and I do not consider it in order until it takes a hold upon the skin of the finger with the slightest touch. It is of great importance in these minute dissections that the very point of the needle should be as slender and sharp as it can possibly be made, in order to penetrate between the delicate elements of the nervous tissue, which by the pressure of a dull point would be torn and destroyed. In dissecting the nervous matter with the view of isolating the ganglionic bodies with their processes, the tissues should, by the most delicate manipulation of the points of the needles, be loosened, avoiding at the same time all tearing, and also too great a pressure of the points upon the glass slide. As I always observe these rules in my labors, I obtained from the tissues prepared by Gerlach's method some of the most beautiful preparations of isolated ganglionic bodies I have ever made. Fig. 2 is a true copy of one of these bodies. As will be observed in this drawing, some of the processes have been torn at a certain distance from their origin, while others have been successfully separated from the surrounding nervous elements, without injury, down to their finest ramifications. As regards their coloring, it is worthy of note that it becomes fainter with the decrease of the diameter of the ramifications. Thus, while the nucleus is colored most intensely, and the ganglionic body with its larger processes presents the brilliant color of the carmine, the subordinate branches become fainter in color in proportion to their decrease in diameter, until the finest ramifications appear only colored pink. This difference in color, however, is only apparent, and is not owing to a greater power of absorption which the larger processes possess in preference to the smaller. On the contrary, it only depends on the mass of the fibrillæ. The greater the mass of the fibrillæ the more, with the exception of the red ray, are they able to absorb the other rays of light. Thus, in cases where a portion of the sheath of a process has been torn away, and the individual fibrillæ, of which the process is composed, separated, as is seen in Fig. 4, it will be found that while the uninjured portion of the process presents the perfect color of the carmine, the isolated fibrillæ appear only pink.

Besides these finest ramifications still in union with their parent processes, a number of others were met with in every preparation made; but, as regards the fine net-work in which they are said to terminate, no trace of it could be found. The same results I found in examining new preparations which I successively made for five days. The only difference observed was a deeper coloring of the whole mass, owing to the extension of the coloring matter into the interstices of the granular substance and the neuroglia. The results, therefore, which I obtained from these examinations made on specimens of grey matter of the spinal marrow, conscientiously prepared after Gerlach's own formula, were the same as I obtained from my examinations of uncolored chromic acid preparations a number of years ago. And as the preparations under discussion were made with the greatest care and accuracy, and examined with superior objectives, made by Mr. Tolles, I feel conscientiously bound to deny the existence of a fine nervous net-work in the grey matter of the spinal marrow, such as Gerlach has described, in which the finest ramifications of the processes of the ganglionic bodies terminate, and from which nerve fibres arise.

Nevertheless, there exists a net-work in the grey matter of the spinal marrow, but only in appearance. It is that representing the delicate connective tissue, pervading throughout the substance of the cerebro-spinal axis, the neuroglia; it is the same apparent net-work which has repeatedly deceived me when I was searching for a true nervous net-work. The very fine fibrillæ of the neuroglia, decussating with each other, present, indeed, the appearance of a delicate net-work with rhomboidal or irregularly shaped meshes when examined in a section or fragment of grey matter. This apparent net-work, however, becomes unraveled as soon as the fragment is carefully dissected into its component anatomical elements, as above described. The fibrillæ of the neuroglia will then appear of a quite considerable length, showing that they never formed part of a net-work of fine regular meshes. The appearance of the neuroglia in the form of a net-work with meshes of a rhomboidal or otherwise irregular form, caused by the decussation of its fibrillæ, is sometimes very striking, and may deceive. I would, therefore, not entirely deny its reticular character, as it is possible that it really represents a delicate net-work of fine elastic tissue fibrils with large meshes. That the neuroglia belongs to this class of connective tissue, is rendered more probable by its immunity from the action of acetic acid. As Gerlach's drawing of his net-work in Stricker's *Handbuch der Gewebelehre*, etc., resembles in some measure the apparent net-work of the neuroglia, I am inclined to think that he mistook the latter for the former, an error which I was very near committing myself, as stated before. The fine fibrillæ of the neuroglia may be easily distinguished by their remaining uncolored. They present a colored appearance only when entangled in the granular substance, but whenever they are examined isolated they at once appear uncolored, for they do not absorb the coloring matter.

If, then, the ultimate ramifications of the processes in question do not terminate in a fine nervous net-work, the question remains to be answered—in what manner do they terminate, and, at the same time, establish a communication between the ganglionic bodies in the grey matter? I have suggested once before that most probably a communication may exist in the form of anastomoses, or, in other words, of a net-work of large and irregular meshes. Though I cannot adduce sufficient evidence to support this suggestion, I have, nevertheless, met with fragments of anastomosing fibrillæ in a few instances, such as are represented in Fig. 5. The fibrillæ forming the anastomosis were exceedingly fine, about from $\frac{1}{700}$ to $\frac{1}{800}$ mm. in diameter; they appear uncolored, and a number of fine granules were observed to adhere to them. There is no doubt but that they represented either portions of the neuroglia or portions of anastomosing nervous fibrillæ. To which order of fibrillæ they really did belong, farther investigations will decide.

In the foregoing pages I have endeavored to show that the ganglionic bodies consist of a number of fibrillæ embracing a nucleus; in other words, that they represent a plexus formed by a greater or smaller number of fine, granular, nervous fibrillæ around a pre-existing nucleus of a complex nature. As there exists no direct connection between the fibrillæ and the nucleus, they represent two distinct anatomical and, probably also, physiological elements of the ganglionic body. And this seems to be the rule throughout the whole nervous apparatus without regard to the mutual arrangement of the fibrillæ themselves; that is, whether they simply pass over the nucleus while lying parallel to each other, or whether they embrace this body in the form of a plexus, or even whether they surround it in the form of a net-work. In all cases we meet with fibrillæ surrounding a nucleus, and in order to show that this is a rule in the construction of ganglionic bodies, I shall now describe the manner in which these bodies are formed in the cerebro-spinal axis of a certain animal belonging to the lower orders of vertebrata, and already known for the enormous size of its colored blood corpuscles and other peculiarities in its organism. I refer to the *Amphiuma tridactylum*, vulgarly called "congo eel" or "congo snake" by the people of New Orleans and vicinity. Several years ago, in a foot-note to my paper "On the Construction of the Dark-Bordered Nerve Fibre," I very briefly alluded to the construction of the ganglionic bodies of the spinal marrow of this animal, stating that they consisted only of a large nucleus over which a few single fibrillæ passed to form a process or axis cylinder. The examinations upon which this statement was based, had been made only in a superficial manner after the paper was already written, and on specimens of spinal marrow which had been lying in a solution of chromic acid for some time. Since that time I have examined the subject more thoroughly, and have been surprised by the simplicity of construction of the ganglionic elements in this animal. I shall begin with those of the spinal marrow.

In the Amphiuma tridactylum, the ganglionic bodies of the spinal marrow consist of large nuclei, surrounded by a network of short granular fibrillæ. These nuclei appear to consist of a dense protoplasm, of considerable refractive power, which in most instances interferes with the examination of the nucleolus within. In the fresh nucleus, therefore, the nucleolus is not often seen, unless by the action of acetic acid. The nucleus (Fig. 6, *a*) presents a homogeneous, slightly greenish appearance, and is bordered by a clear double contour. The nucleolus also shows a double contour, and one or two small granules within its centre; its diameter measures about $\frac{1}{150}$ mm. The net-work closely surrounds the nucleus, and its meshes, together with the granular nature of its component fibrillæ, can be distinctly recognized in the fresh specimen. From the net-work a few fibrillæ are seen to arise, which, by converging toward each other, form a small axis cylinder process (Fig. 6, *b*). Some other fibrillæ, also, arising from the net-work, pass to neighboring ganglionic bodies (Figs. 7 and 9).

The form and size of the ganglionic bodies, as may be inferred from the description of the construction just given, depend upon the form and size of the nucleus, which differs very much in different parts of the spinal marrow. In examining a transverse section of the spinal marrow of the amphiuma, it will be found that the distribution of its ganglionic bodies deviates to a certain extent from that usually observed in the higher vertebrata. While in the grey matter of the spinal marrow of mammalia, the ganglionic bodies, especially those of the anterior horns, are mostly arranged in groups, and confined to the grey matter, they form, in the amphiuma, not only the greater part of the whole grey matter, but are found singly distributed throughout the white columns. In the grey matter, they are crowded around the central canal (Fig. 13), forming a ganglionic zone of considerable breadth, from which they extend to form the anterior and posterior horns. The ganglionic zone or ring around the central canal is particularly formed by the smaller bodies, of a more or less roundish form (Fig. 9), measuring from $\frac{1}{50}$ to $\frac{1}{30}$ mm. diameter. Extending from this zone into the anterior and posterior horns, the ganglionic bodies increasing in size, assume a more oblong form, and are placed in such a manner that their long axis is more or less parallel with the radius of the section of spinal marrow. Thus, the largest ganglionic bodies (Fig. 13) are found at the periphery of the grey matter, extending into the white columns. This arrangement, however, is only a general one, for there are many exceptions met with, in which smaller ganglionic bodies are placed near the periphery, or, also, in the white columns. The long diameter of the oblong ganglionic bodies ranges from $\frac{1}{40}$ to $\frac{1}{20}$ mm.

The ganglionic bodies forming the zone around the central canal, are mostly of the smaller kind. They are placed close to each other, and, as it appears, communicate by means of the fibrillar network surrounding the nuclei. From the network of a number of these bodies, a process consisting of only one fibril is seen to arise. Whether this process passes at once to the periphery of the grey matter, to contribute to the direct formation of axis cylinders, I cannot say; but it is certain that filaments arising from the net-work of the individual ganglionic bodies anastomose with each other, as seen in Fig. 9; and, also, that from these anastomoses, independent nervous fibrillæ arise, which, during their course to the periphery, join, in order to form axis cylinders. From the net-work of the larger ganglionic bodies, distinct processes, consisting of two or three fibrillæ, are seen to arise (Figs. 10 and 11). These processes, the course of which I have been able to trace to a considerable distance, and in a direct line toward the white columns, represent distinct axis cylinders. From the largest ganglionic bodies, met with at the periphery of the grey matter, especially in the anterior horns, processes of considerable size are seen to arise. The fibrillæ of these processes, however, are not all derived from the net-work of the body from which the process apparently arises; but, on the contrary, the process is formed by additional processes, or axis cylinders, coming from other parts, and surrounding the ganglionic body in question, before joining its process, as seen in Fig. 11. In these instances, the fibrillæ appear to be imbedded into an inter-fibrillous substance, which connects even a number of these larger bodies into one general mass. Considerable portions of this inter-fibrillous substance are frequently found adhering to the larger ganglionic bodies.

The delicate net-work surrounding the nuclei consists, as already stated, of very fine fibrillæ. In the fresh specimens, especially, the individual granules, of which these fibrillæ are originally formed, can be distinctly seen. The meshes formed by these short and granular fibrillæ are of an irregular polygonal form. In accordance with the form of the nucleus, which they surround, they appear either more round or oblong. In many instances, small, dark looking granules are observed at the angles of the meshes. Besides the communication formed between the net-works of neighboring ganglionic bodies by those anastomosing filaments already described (Fig. 9), the neighboring net-works seem to be furthermore connected by their meshes. Thus, in dissected preparations, a number of ganglionic bodies are met with, upon which the net-work does not pass to the very margin, because here it has been torn from the net-work of the neighboring ganglionic body by the manipulation with the needles. In chromic acid preparations, this phenomenon is especially observed, on account of the remaining portion of the net-work contracting by the action of the acid. For this reason, the specimen should not have remained too long in such a solution. The best preparations are obtained by putting the spinal marrow, directly after its removal from the animal, into a very weak solution of bichromate of potassa, in which it is left for twenty-four, or even forty-eight hours, and then transferred to the carmine solution. Being prepared in this manner, small portions of grey matter, distinguished by a more intense coloring, are dissected by means of fine needles. If the preparation remains longer in the solution, the net-work becomes too hard, causing the breaking of the communicating fibrillæ. I have succeeded in making thin sections of spinal marrow sufficiently hardened for the purpose, which, after being colored, were mounted in a solution of gum dammar. The highly refractive power of the colored nuclei, however, interfering with the rays of light proceeding from the net-work, deprives these preparations of their usefulness. For this reason, glycerine has proved to be the best medium for the study of the more intimate relations of the nervous elements concerned.

The arrangement of the nervous elements in the cerebrum of the Amphiuma is still more striking than the one in the spinal marrow just described. The whole nervous substance here seems to consist of a continuous net-work, with small regularly formed meshes in which the nuclei are literally imbedded. From the delicate meshes of this net-work, especially in the vicinity of the nuclei, larger meshes, consisting of coarser fibrillæ, are arising to give origin to those fibrillæ forming the axis cylinders of the nerve fibres (Fig. 14). The nuclei are in every respect of the same nature as those in the spinal marrow, already described, with the exception of their form, which is more or less round. In thin transverse vertical sections of the cerebrum, large accumulations of nuclei are observed to form the surface of the ventricles. They appear to represent the larger cerebral ganglia, the corpora striata, thalami optici, etc., and it is especially in the vicinity of these masses that the greater portion of axis cylinder fibrillæ are seen to arise. Properly speaking, there is no distinct ganglionic cortical layer of the cerebrum, for the nuclei representing this layer are comparatively few in number, and singly scattered throughout the general net-work, without any apparent system or order.

The arrangement of the nervous anatomical elements in the cerebro-spinal axis of the Amphiuma, being more simple in form than in animals of a higher order, shows again most distinctly the individuality of the fibrillar or conducting element, opposite the nuclei. Before drawing any general conclusions, however, from the facts presented, we will take a glance at the structure of the ganglionic bodies met with in the ganglia of a still inferior order of animals, namely, the insects.

The ganglionic bodies found in the ganglia, representing the nervous centres of insects (Figs. 15 and 16), apparently consist of a mass of granular protoplasm, inclosing a large clear nucleus of about $\frac{2}{75}$ mm. in diameter. This nucleus, the vesicular nature of which is indicated by a fine double contour, contains a nucleolus of about $\frac{1}{200}$ mm. in diameter, which, however, is composed of three large granules of a greenish lustre, apparently closely adhering to each other (Fig. 15). Whether the granules of the protoplasmic mass surrounding the nucleus are regularly arranged in fibrillar rows, cannot be asserted; but it is certain, that from this mass a great number of fine short fibrillæ arise, which, to some extent, anastomose with each other. From these anastomoses a considerable number of long delicate fibrillæ arise, by the junction of which a large process is formed. The anastomosing filaments themselves extend to neighboring ganglionic bodies, stablishing a mutual communication of a number of these bodies, forming a group. The large processes arising from these ganglionic bodies join to form the large nervous trunks which leave the ganglion.

The arrangement of the nervous elements in the insect, resembles in type, as we see, that in the nervous system of the Amphiuma, above described. In both instances, the communication existing between the individual ganglionic bodies is more direct and extensive than in animals of a higher order. In accordance with this arrangement the impression made by peripheral stimuli upon these centres, as well as the nervous energy emanating from them, must be more diffused; the supply of energy to the various organs of these animals, therefore, cannot be as nicely distributed as in animals of a higher organization, in which the nervous elements are differentiated from each other to a greater extent.

The conclusions which may be, finally, drawn from the facts presented in the preceding pages are, that all ganglionic bodies or ganglionic cells, are composed of two elements, anatomically and functionally distinct from each other. The one which is represented by fine nervous fibrillæ, undoubtedly serves to conduct nervous energy, liberated by stimuli applied at the peripheral surface, or emanating from a centre. The function of the other element, represented by a vesicle of a complex nature, the nucleus, is more difficult to determine; but is probably related to the liberation of potential energy residing in it. In supposing that the energy liberated by a stimulus upon the sentient surface may be, without passing through a nerve centre, directly transmitted through the nervous fibrillæ to a muscle or a gland, it might be expected that a contraction of the muscle or a flow of secretion from the gland was to take place, provided the stimulus were sufficiently intense for the purpose. And though such an occurrence may appear improbable, it is not proved that in some instances nervous action may not take place in this manner; for, as we have seen, a number of fibrillæ arriving from the ganglionic cell through one process, leave by way of another neighboring one, without having ever approached the nucleus occupying the centre of the whole. The greater portion of the fibrillæ passes, nevertheless, close to the nucleus, or even comes into actual contact with it. The inference, now, which may be drawn from the relative positions of the fibrillæ and nucleus, is that the nervous stimulus, traveling along the former, becomes increased in strength by an additional amount of liberated potential energy, residing in the latter. This hypothesis is not altogether bare of foundation, when the complex constitution of the nucleus is taken into account. The interior of the nucleus is, besides containing the nucleolus, closely packed with minute granules, and the nucleolus, also of a vesicular nature, is not only similarly filled with granules, but moreover contains one or two bodies of an ambiguous nature, which manifest by their unusual reddish lustre, a considerable power of refraction. The presence of these bodies in the interior of a nucleolus, without regard to their nature, is very unusual; so much the more as one of them contains, even, a dark granule in its centre. Thus, passing from the outer surface of the nucleus toward its centre, we successively meet, in the first place, with the membrane of the nucleus and the granules in its interior; next, with the membrane and granules of the nucleolus; then, with the vesicles or vacuoles in the interior of the latter, and, lastly, with the dark granule in the centre of one of the vacuoles. It seems obvious that nature should have assigned a particular function to a body of a constitution as complicated as that of the so-called "nucleus of the ganglion cell;" and I cannot forbear to point again to the fact, that if any part of the ganglionic body, or ganglionic cell, deserves the name of a nervous cell at all, it is the nucleus; for it alone represents an organic cell in the true sense of the word. Though we know nothing of the material interchanges going on between the different morphological constituents of this cell, we may well presume that its function is related to the secretion, or in other words, generation of nervous energy. This energy may, in its potential form, remain stored up within its particular organism, the nervous cell, to be liberated by the nervous stimuli passing along the nervous fibrillæ surrounding it. Thus, the ganglionic bodies must be looked upon as minute depots of nervous energy, destined to impart to the passing nervous current a greater intensity, by an additional amount of potential energy, which their nuclei, or true nerve cells, contain, and which is liberated by the stimuli passing along the nervous fibrillæ. The whole phenomenon may be compared with that of electric induction.

There can be no doubt but that the office of the nuclei of all tissues is an important one. I, for my part, have always regarded these bodies as the direct descendants of the cells, resulting from the segmentation of the egg, and, as the special organs through which the characters and peculiarities of the parent are transmitted to the offspring; and, therefore, I also presume, that the differentiation of the tissues during the earlier periods of embryonic life, and the preservation of their particular form and character in extra-uterine life, depends upon the influence exerted upon them by the special nuclei each tissue contains. That the integrity of the tissue depends upon the integrity of its nuclei, is a pathological fact. To corroborate this view, I need only refer to the degeneration of the minute blood-vessels, commencing in pathological changes of their nuclei, as an example. And it is for this reason that I venture to direct the attention of neurologists to the condition of the nuclei of the ganglionic bodies in diseased nervous tissues, constantly met with in organic diseases of the nervous system.

As may be presumed from what I have said above, I consider the ganglionic body as an organ destined for the generation and distribution of nervous energy; but incapable of receiving sentient or conscious impressions. It is obvious that impressions can only result in consciousness in those nervous elements which represent the terminus of the nervous apparatus, as well as its commencement in the cortical layer of the brain. Though a large number of ganglionic bodies is distributed throughout this layer, they cannot be looked upon as elements, capable of receiving the manifold sentient impressions coming from the periphery, unless the nucleus is regarded as the particular receiving organ. Such a supposition, however, is not logical for the reason that the nervous fibrillæ, carrying the stimulus to the centres, do not terminate in the nucleus, as has once been asserted by some histologists, but, on the contrary, pass it without entering into any anatomical communication with it, and, finally, terminate in a delicate net-work consisting of minute meshes, formed by minute granulous fibrillæ, and extending throughout the whole cortical layer. The interspaces left by the meshes are filled up by a substance consisting of minute granules, similar in character, or even perhaps identical with those granules of which the delicate net-work is formed. Looking now upon the subject in the true light, it will appear more probable that the stimulus coming from the sentient surface of the periphery, after being increased in strength by an additional amount of liberated potential energy from the true nervous cell, viz.: the nucleus of the ganglion cell, passes along the nervous fibrillæ surrounding this body, without interruption, and, finally, arrives in the terminal nervous net-work, to be, in proportion to its strength or intensity, diffused throughout its delicate meshes. But, as the granules of which the fibrillæ forming the meshes of the net-work are composed, are very probably identical with those representing the substance filling up the meshes of the net-work, it may be also presumed that the stimulus does not remain confined to the fibrillæ of the net-work, but, moreover, extends to the granular substance itself. Thus, the delicate fibrillous net-work, together with the granular substance filling up its meshes, both forming the greater portion of the cortical layer of the brain, would represent the special element, capable of transforming impressions, received from the sentient periphery, into consciousness.

In support of this view, I will mention only one of the many facts which may be adduced in proof of the fallibility of the theory, which locates consciousness in the ganglionic cells of the cortical layer. In admitting as a fact, that the image of external objects is received upon the retina in the same manner as upon the glass plate in the camera of the photographer, it is, in order that this retinal image may be perceived by the mind, absolutely necessary that the impressions upon the retina by each of the countless rays of light, of which this image, in its various colors and shades, is composed, should be carried and imprinted upon the nervous substance of the cortical layer. And, moreover, the mutual arrangement of the impressions thus made upon the cortical layer, and resulting in the perception of the image by the mind, should exactly correspond to the mutual arrangement of the rays of light emanating from the original object. Such an exact arrangement of the impressions upon the cortical layer, however, could not be obtained, if the ganglionic bodies of this layer were the only receiving organs of peripheral stimuli, because the mutual arrangement of these bodies themselves is very irregular. It is true that they are, to a certain extent, arranged into different strata, and that they differ comparatively in size and form in each stratum. Notwithstanding the size, form and mutual arrangement, even in each individual stratum, is too irregular to receive the impressions made by the mathematically arranged rays of light upon the mathematically arranged peripheral elements of the retina, and transmitted through the fibres of the optic nerves to the cortical layer. If we, on the other hand, regard the granular substance of this layer as the receiving organ, the whole process is easily explained, for the number of granules of this substance is so great, and their arrangements so close and regular, as to offer to the stimuli of light a suitable substance of sufficient extent to receive the individual impressions of all the rays, transmitted by each individual nervous fibrilla. In this manner alone, it appears possible that the cerebral image of the object may be a true copy of the retinal image, produced by the stimulus of the rays of light, emanating directly from the object.

The analogous arrangement of the anatomical parts of other systems in the organism, also corroborate this view. If we look, for instance, at the circulatory apparatus of the blood, we find that the arteries and veins are but simple organs of conduction, in which the blood undergoes no perceptible change, as the material interchanges between this fluid and the tissues are taking place while it passes through the capillaries, a system of minute vessels arranged in the form of a net-work. The continuous current of the blood in the arteries, when arriving at the capillary net-work, is broken, its force becoming distributed and diffused throughout the capillary channels in a slower manner; and, the pressure exerted upon the blood being extended in more than one direction, affords an opportunity to this fluid to carry on its material interchanges through the delicate walls of the capillaries with the surrounding tissues.

In an analogous manner, the nervous current, passing along the fibrillæ of the axis cylinders and ganglionic bodies, becomes diffused throughout the terminal net-work of the cortical layer, and leaves its impressions upon the granular substance to which it is communicated.

In the grey matter of the spinal marrow, medulla oblongata, pons varolii and the cerebral ganglia, we also meet with the granular substance, filling up the interspaces left, though not to so large an extent as in the cortical layer of the brain. But, as the meshes of the anastomoses probably formed by the finest ramifications of the processes of the ganglionic bodies must be quite large, the nervous stimulus is not as minutely and regularly diffused throughout, as well as communicated to the granular substance; and, in consequence, the impressions made upon this substance cannot be as distinctly defined, as in the cortical layer of the brain. Judging, however, from the arrangement of the terminal elements in the grey matter of the spinal marrow, medulla oblongata and other nerve centres, being analogous to that of those elements in the cortical layer of the brain, it seems reasonable to suppose that these parts, also, should be the seat of a limited amount of consciousness, which, though insufficient for intellectual manifestations, may nevertheless suffice to give rise to those well known reflex and automatic actions.

EXPLANATION OF THE ILLUSTRATIONS.

Fig. 1.—Ganglionic body of the spinal marrow of the calf, showing its structure of granulous fibrillæ. Magnified 420 diameters.

Fig. 2.—Ganglionic body of the same locality, isolated by Gerlach's method. Magnified 275 diameters.

Fig. 3.—Section of a ganglionic body, showing the internal arrangement of its fibrillæ. Magnified 420 diameters.

Fig. 4.—Fragment of a process of a ganglionic body, with a portion of its sheath removed, and showing its fibrillæ isolated. Magnified 420 diameters.

Fig. 5.—Anastomosis of filaments. Magnified 420 diameters.

Figs. 6 to 12.—Ganglionic bodies of the spinal marrow of *Amphiuma tridactylum* (Fig. 6, *a*, free nucleus). Magnified 420 diameters.

Fig. 13.—Transverse section of spinal marrow of the same animal. Magnified 50 diameters.

Plate I

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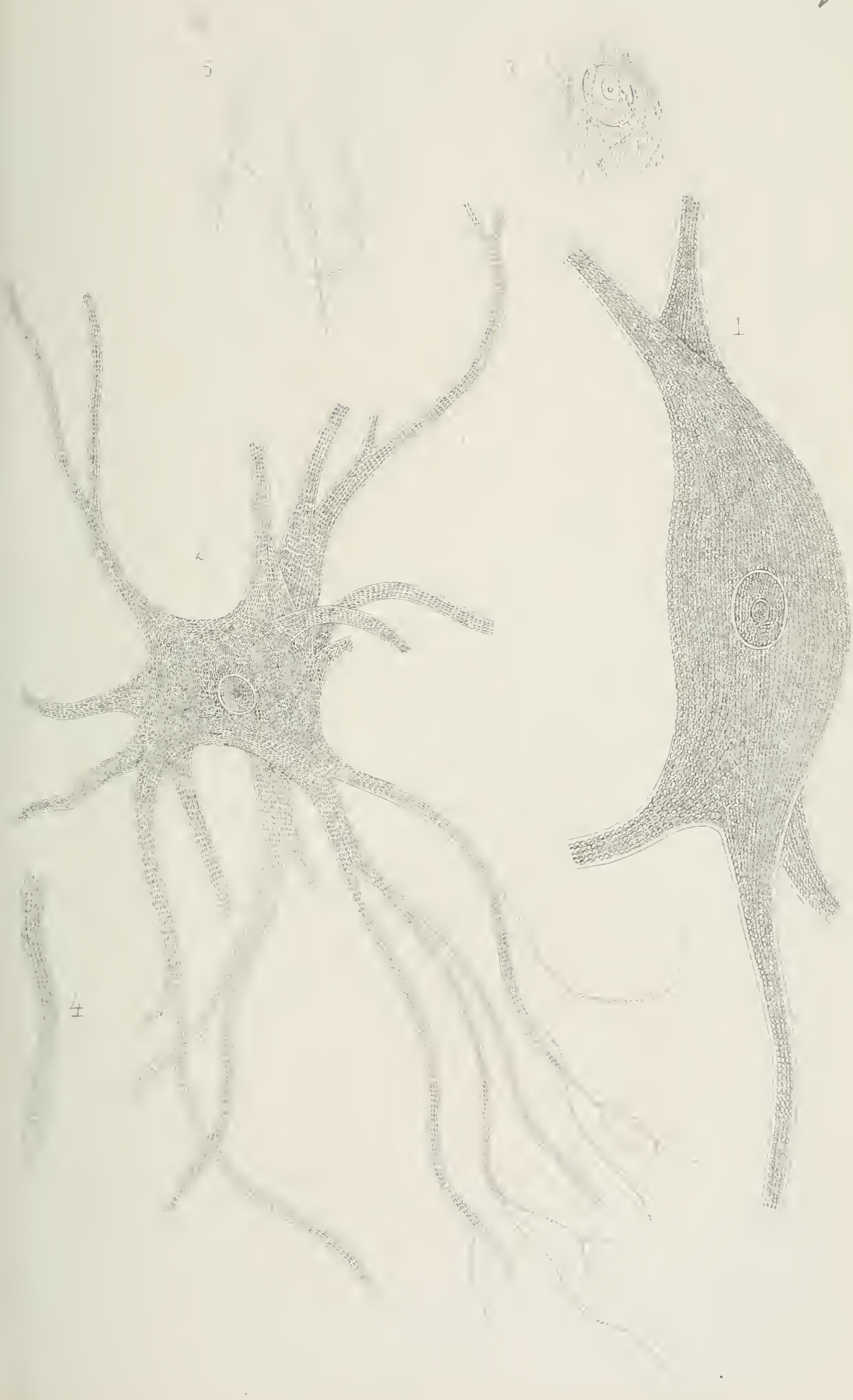

Plate II 262

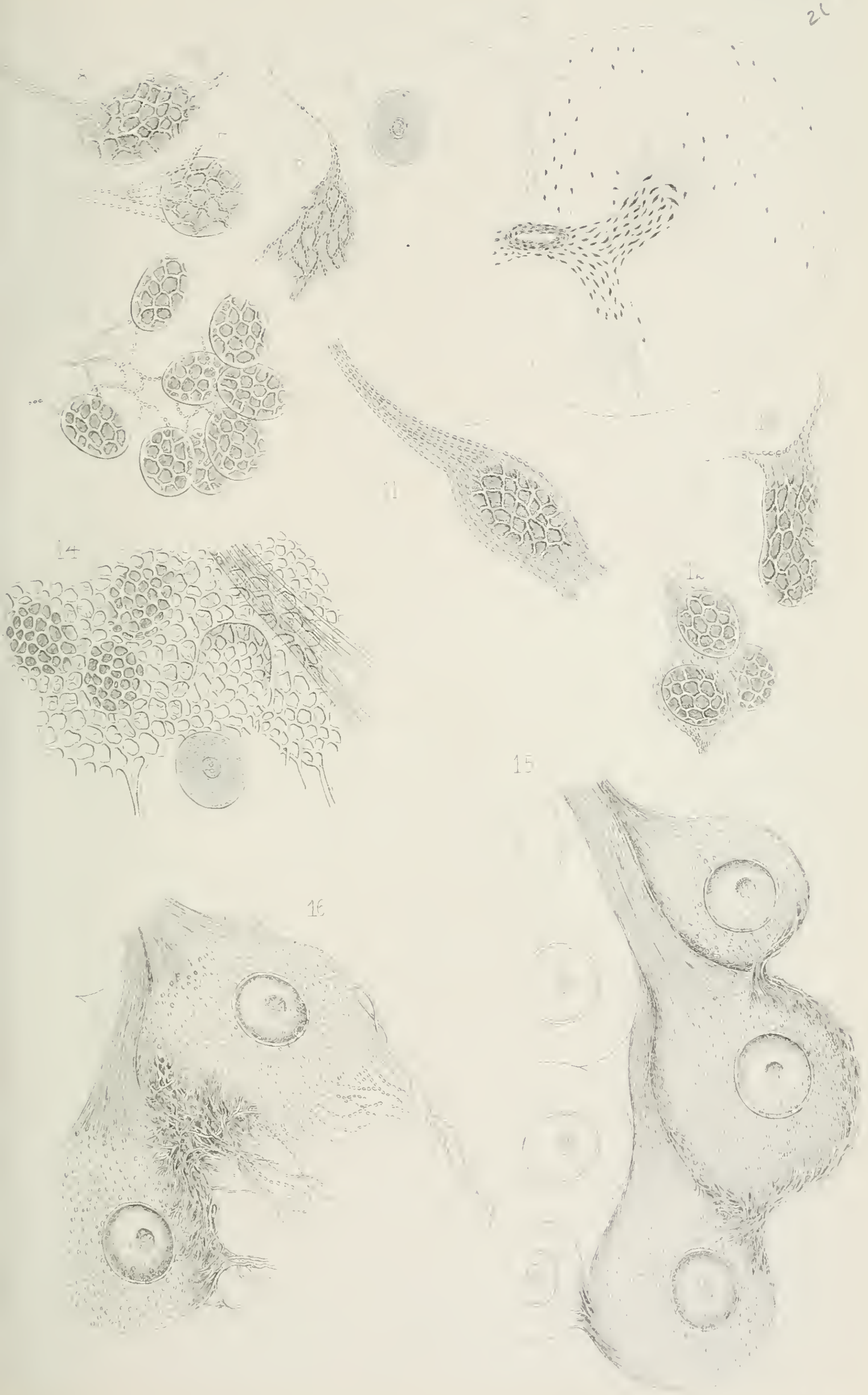

Fig. 14.—Ganglionic bodies with their net-work from the cerebrum of Amphiuma tridactylum. Magnified 420 diameters.

Fig. 15.—Ganglionic bodies from one of the abdominal ganglia of *Blatta orientalis* (cockroach), showing their mutual connection by means of anastomosing nervous filaments.

Fig. 16.—The same, after the action of acetic acid.

Art. II.—CONTRIBUTIONS TO ENCEPHALIC ANATOMY.

By Edward C. Spitzka, M. D., of New York.

PART V.

The Organology of the Island of Reil.

IN previous pages of this series I have adverted to the fact that an anatomical identity or similarity existing between the corresponding areas of two given brains, does not imply that those areas are also functionally homologous. The less likely is an anatomical similarity to involve a physiological similarity, the nearer we approach the highest centres ; while an atrophy (I am speaking of the *normal* atrophies in the zoological series) or over-development of the central tubular grey matter and the nerve roots, can be readily referred to corresponding defects or higher developments of the related periphery, and comparative physiological deductions can be derived from this relation ; the cerebral and cerebellar cortical areas do not admit of such being made. I consider the establishment of this fact so important in its bearing on the question of cerebral localizations, and especially on those which are grounded on localized irritations of the cortex in the lower animals, that I have made an extensive series of dissections of that cortical territory which can be most readily identified in the amniote animals, namely, the island of Reil, in order to illustrate the principle involved.

Various views have been advanced by morphologists and embryologists as to the cause of the cortical retraction, which results in the more or less complete isolation of the island.

Some of these are fanciful, and I shall not refer to them in detail. I find that those animals which in the adult condition exhibit a rudimentary *fovea* corresponding to the *fossa Sylvii*, while in their embryonic state undergo a marked prosencephalic incurvation. No animal possesses this fossa, unless it undergoes this incurvation; for this reason, and for others to be detailed, I regard the primary cause of the development of the fossa to lie in the same traction which determines the prosencephalic curve. This traction is exercised by that portion of the *hypoblast* which furnishes the future pharynx, and which in the early embryo is adherent to the base of the grey matter surrounding the third ventricle. The resulting tension is transmitted to that part of the hemisphere which is most intimately connected with the point where the hypoblast is adherent, and it thence results that only a basi-lateral part of the thin-walled hemispheric vesicle is drawn in. Even the brain of the adult human being exhibits a direct continuity of the lower apex of the island with the grey, perforated lamina, which is continuous with the basilar grey matter of the third ventricle, in other words, with the infundibulum of the pituitary body. We know that the latter is a process dragged out of the base of the brain when the traction of the hypoblast reaches its maximum. At the floor of the ventricle, corresponding to the furrow of traction, there must exist a corresponding projection inwards, and this projection is the starting-point for the development of the corpus striatum.

We thus perceive that the traction* exercised by the hypoblast upon the central nervous system produced, firstly, the prosencephalic curve; secondly, the fovea of the Sylvian fossa, and through the latter, the corpus striatum; finally, the hypophysis cerebri (pituitary body). Those observers who, like His, refer the island of Reil to the checked expansion of that cortical area which, from its vicinity to the corpus striatum, becomes fused with it, have reversed the true relation of cause and effect; it is not the position of the corpus striatum which determines the formation of the Sylvian fossa, but the latter

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*Due, in its turn, to the discrepancy of growth between nervous blastema and hypoblast, greater in higher than in lower animals, and absolutely *nil* in amphioxus.

which determines the former. If it were as His claims, then the outline of the fossa, respectively the island, would correspond to, and be determined by the corpus striatum, and the deepest part of the fossa would correspond to the thickest part of the latter ganglion; but this is not so; the island in lower animals does not follow the outline of the latter, and as to the second point, it is controverted by the brain of the turtle, which has a shallow Sylvian fovea whose deepest part corresponds to a depression of the corpus striatum. It is true, however, that all animals that possess a true corpus striatum, also have a Sylvian depression, and that those having the latter, also possess the former, but this is explained equally by the representation made above, as by the hypothesis of His.

Since there is a great deal of ambiguity in the use of the terms fissure of Sylvius, island of Reil, and fossa Sylvii, I shall venture to suggest a uniform, and I trust, applicable nomenclature. It is obvious that the shallow groove of a turtle's or a serpent's brain, although homologous with the island of Reil, hardly deserves the term "island," nor even that of a fossa. I propose to call such depression of the cortex "fovea centralis." Such a fovea is present in all the true reptiles and in birds. A higher degree of development is manifested where a more or less sharply defined area is drawn in, lying at a deeper level than the surrounding cortex; let us term this "fossa centralis;" such a one is present in the rodentia, insectivora and some marsupials. A still higher stage is that in which the surrounding convolutions more or less completely overlap the retracted area; since the latter, therefore, becomes a specialized cerebral division, and in its rotundity and bulk approaches other demarcated cortical provinces, it now merits the appellation "lobus centralis."* It is found in the carnivora, both terrestrial and aquatic, the solipedes, herbivores, elephant, hippopotamus, old and new world monkeys, and in man. In this series it presents varying degrees of concealment beneath the overlapping margins of contiguous lobes; it therefore admits of further classification into the "concealed" and the "exposed" lobus centralis. It is completely, or almost completely concealed in *man*, the troglodyte apes, the baboon, Cercopithecus macacus, Lagothrix. Cebus and Ateles, also the seal, possibly in the porpoise and very probably in other simian forms not examined by myself.

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*Ecker.

In none of the anamniote animals have I been able to detect anything that could be fairly interpreted as even a "fovea centralis." I was consequently much surprised to find that Meynert, in a recent publication* refers to a specimen of a *fish's* brain, demonstrated to him by my teacher, Professor Shenk, which he claims proved the universal dependency of the island of Reil on the development of the corpus striatum. This was a lamentable misconception, for, in the first place, neither the bony nor the cartilaginous fish possess true lateral ventricles; they have no corpus striatum, nor do they either in the embryonic or mature condition exhibit anything even remotely allied to the Sylvian fossa. In the rest of his monograph. Meynert very properly insists that the fissure of Sylvius should be considered the starting-point for the nomenclature of the gyri, but in the instance alluded to his desire to establish a wide-spread homology has led him into an error; there is a limit beyond which it would be fruitless to attempt homologizing here.

The "lobus centralis" of the higher mammalia follows no definite law visible on first sight, as regards its dimensions or relations. If we were to assert that it was largest in the highest animals, we should be in error, for this lobe is larger in the hippopotamus than in the seal; if we were to determine the position intellectually of an animal by the number of gyri on this lobe, we should be forced to the ridiculous conclusion that the hippopotamus rivaled the human being and far exceeded the anthropoid apes. In like manner, would it be erroneous to state that the more completely the island of Reil (lobus centralis) was covered by surrounding cerebral parts the higher its own development. In the seal, although entirely covered, it is rudimentary; in the human being, in which it is also covered, and in the hippopotamus, in which it is practically uncovered, on the other hand, it is very large. Even

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*"Die Windungen der convexen Oberflaeche des Vorderhirnes bei Menschen, Affen und Raubthieren," von Prof. Theodore Meynert. *Arch. fuer Psychiatrie*, VII.

where in allied groups, the island is entirely covered, the dimensions, as well as the convolutions of the latter, may vary greatly, as instanced in the anthropoid and lower apes compared with man.

We thus see that no constant relation exists between the degree of development of the lobus centralis and that of the remainder of the cortex, from which it follows that it is no indication of cerebral development in general.

It now remains for us to see whether there is any constant agreement between the island and the fibre masses, or ganglia, at the base of the brain. In regard to this question it is unfortunate that we have not a sufficient number of observations at our disposal. I especially feel the absence of such dissection in the case of the seal* and hippopotamus.† I limit myself, therefore, to the rodentia, terrestrial carnivora, cheiroptera, and the simians, including man; in this series, I find a relative agreement between the outline and dimensions of the island of Reil and the nucleus lenticularis of the corpus striatum.

Since the island and the bordering areas send their fibres into this ganglion, the cause of this dependency is clear. According to Meynert, the efferent fibres of a large portion of the nucleus lenticularis run inwards, as the fourth stratum of the ansa peduncularis, to become the most internal fibres of the pes pedunculi: these same fibres are supposed on very strong grounds to place the cranial nerve nuclei under the control of the higher centres. We judge the latter from the course of the raphe fibres, and the greater thickness of the raphe in those regions where the hypoglossal, facial and motor trigeminal nuclei are situated. I can attribute no value to the contradictions of Flechsig,‡ who, misled by his exclusive study of a single method, maintains that the entire mass of the pyramidal fibres contained in the pes pedunculi terminates in the præ- and post-central gyri! A large portion terminates thus undoubtedly, but not the whole. In the light of Meynert's description, there would exist a communication between the cortex of the island and the cranial nerve nuclei. We would except from this the nuclei of the oculo-motor, trochlearis and abducens nerves, in whose case other relations can be established, while no raphe fibres can be traced to them. This connection finds a strong support in pathological experience, and receives confirmatory support from comparative anatomy. The seal, whose facial muscles are rudimentary and whose tongue is not freely movable, has the most reduced island of Reil in the mammalian series, and as the sequel will show, it is doubtful whether its insula is strictly homologous with that of other mammals.

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*The only specimen of the former in my possession decayed in the preserving fluid.

†Examination not concluded at date.

‡"Weitere Beobachtungen ueber den Faserverlauf innerhalb der nervoesen Centralorgane." *Centralblatt*, No. 3, 1877.

I shall now proceed to describe the lobus centralis in a series of higher mammals in order to illustrate the propositions advanced, and to determine the much-vexed question of the localization of speech.

In the land carnivores, such as the cat, lion, and dog, the lobus centralis runs anteriorly into the orbital surface of the frontal lobe without any boundary, only a small triangular part of the island is covered, and this is perfectly smooth, presenting not even the faintest traces of sulci or gyri, the nucleus lenticularis, which in these animals is very small compared with the corpus striatum, corresponds pretty accurately in its outline with the covered part of the lobus. The bear, whose brain in all respects far exceeds in development that of other land carnivores,* presents a marked advance in these respects; not only is his insula larger, but it actually possesses a single sulcus running in an antero-posterior direction and dividing it into two gyri. Its anterior border is externally as vaguely defined as in the lion or dog, but the lenticular nucleus is larger proportionately, and extends further forwards than in these animals. As the bear's brain presents a marked advance on the other carnivora, both in the general character of the brain and the development of the special territory in question, the seal exhibits a still greater development of the brain in general, but a remarkable retrogression of the lobus centralis. If after identifying the deep and diagonally running fissure of Sylvius from among the numerous meandering sulci of the seal's cerebrum, we separate its walls, we shall, after tracing the fissure to a greater depth than even in the human brain, be surprised to find but a narrow area, measuring in diameter not quite three-quarters of an inch, representing the lobus centralis. This area, however, is not overhung as in other placental mammals by the parts analogous to the operculum, and posteriorly and upwards the area representing the lobus centralis, after dividing into two gyri, runs out of the fissure of Sylvius to gain the free surface of the convexity. Its two sulci run backwards and inwards on either side of that fissure which corresponds to the fissure of Rolando.

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*As shown in the size and convolution of the hemispheres, the posterior inflection upwards of the gyrus fornicatus, the high development of the frontal and temporal lobes, the prominence of the olivary bodies, and the almost complete concealment of the trapezium.

At the base of the hemisphere, the apex to which this area dwindles is continuous, like the lobus centralis of other animals, with the lamina perforata antica; but in view of the fact that it is not overhung by the operculum, but continuous with the præcentral and post-central gyri, it is to be doubted whether we have here the true lobus centralis to deal with. I have usually been able, where other criteria failed, to utilize the extent of the claustrum as a gauge for the proper extent of the insula, but in the instance of the seal, circumstances adverted to prevented this. It is certainly suggestive that while the seal, with its greater general intelligence, should possess a cerebrum much more intricately convoluted than the bear or even the anthropoid apes, the latter animals should far exceed it in the development of the island. The localization of the more delicate motor innervations of the tongue and facial muscles gains some support from the fact that the seal's facial muscles are rudimentary and the tongue movements limited, while the facial movements of the monkey tribe are various and complex. In the bear also the tongue is an important organ of prehension, and can be employed outside of the mouth with greater skill than the paws. I presume that the otter, which in its anatomical structure exhibits a transition from the land to the water carnivora, will show peculiarities of its cerebral structure calculated to elucidate that of the seal.

In monkeys we find every transition, from the smooth and limited island of the smaller species to the extensive and richly convoluted island of man. As we rise from the lower to the higher, we observe that while this area is triangular in all, that the altitude of the anterior vertical side of the triangle shows the chief increase: so that while this region is a very acute triangle with its sharper apex directed backwards in lower monkeys, it has almost become isosceles in man. The horizontal diameter shows the least increase. In the cercopithecus, cebus, lagothrix, macacus, and ateles, the lobus centralis is smooth and differs merely in size; in the inferiorly organized platyrrhine monkeys, the anterior inferior border is frequently uncovered.

In the baboon we already perceive a distinct sulcus, which, like that of the bear, runs antero-posteriorly; in addition there is a faint indication of another one, parallel to and above it, corresponding to the middle height of the overlapping operculum. The orang-outang shows two clear sulci running as from a common inferior starting-point and separating above, thus indicating the course of the sulci in man. The orang-outang's island contains, therefore, three gyri. In the chimpanzee the lobus centralis is like that of the orang, completely covered by the operculum, the (third) inferior frontal gyrus and the temporal lobe, the same relation as that obtaining in man. It is more extensive in the chimpanzee than in the other anthropoid, and bears the same relative proportion to the entire cerebral dimensions as in the human being. It possesses four distinct gyri, the last two branching from a common root. In one chimpanzee, whose brain was atypically asymmetrical, I found indications of a fifth gyrus posteriorly, but this is probably an exceptional occurrence, and as I have no record of the condition of the atrophic hemisphere, has no special value.

From the fact that not only the anthropoid apes, but even some Old World monkeys and even the bear, possess gyri on the island, it follows that the categorical dictum, "only man possesses gyri on the island of Reil," must, like Richard Owen's claim that "man alone possessed a posterior cornu, and hippocamp," or that "in man alone the cerebrum overlaps the cerebellum," be relegated to the errors of the past.

In the chimpanzee the gyri of the lobus centralis run rather upwards than backwards, and show, as stated, a tendency to bifurcation, thus increasing the resemblance to the human island.

The latter, however, is far more complex than that of the highest anthropoid (under normal circumstances); it contains not only more gyri, but these are separated by deeper sulci than those in the troglodyte. The usual condition is that from four to six gyri run from the lower angle upwards and bifurcate at the upper half of the island. The more posterior the gyrus the more oblique is its course and the greater the tendency to bifurcation, so that on the subdivisions of the last gyrus a ternary subdivision is occasionally noticed. Since the inner aspect of the operculum is provided with gyri, which constitute an exact negative of the insular gyri, dovetailing with them by immediate contact, it follows that the more highly developed the island, the more highly developed will be the internal face of the operculum. In imbeciles I have frequently found merely five undivided gyri, and in one instance a disposition exactly imitating that of the chimpanzee. All these individuals were possessed of articulate speech, though, corresponding to their general condition, only to a limited extent. In terminal and secondary diminution of the intellect, accompanied by corresponding loss of word-imagery, nothing aberrant could be discovered in the disposition of the gyri; this might of course have been anticipated. But in one instance of congenital imbecility with almost total absence of the faculty of language, the gyri were numerous and well developed, as were all the other convolutions. The brain of this patient, whom I have designated as a macrocephalic idiot, weighed sixty-eight ounces, and is with one possible exception the heaviest human brain on record. It is to be noticed, however, that the neuroglia was hypertrophied, the ependyma layer of the cortex thickened and the peduncular tracts disproportionately small.*

I will not hesitate to predict, judging by analogous cases, that in such an idiot as the well-known John Rouse, of Randall's Island, the island of Reil is far behind that of an anthropoid ape, but yet this remarkable monstrosity has learnt a few words, which he uses with a certain purpose, although in every other respect he seems to be inferior to our ordinary domesticated animals.

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*The weighing of this brain was not undertaken with that care which could enable me to vouch for the exact accuracy of the figure given. I have them at second hand.

In certain idiots, not only has the lobus centralis been found defective, but even uncovered by the parts usually bordering the Sylvian fissure.

And these facts do not disprove the localization of the speech innervations in the island and operculum, but they show that this region may be comparatively well developed and speech be absent (troglodyte), whereas, on the other hand, the partial possession of the speech faculty may be associated with a far more defective island (in case of certain idiots). This shows that other factors must co-operate with the insula in this faculty, and that these lie in the association tracts extending from the island to distant cortical parts; and that these tracts have been ground out in countless generations of beings whose ancestors acquired the faculty *gradatim*, are inferences in perfect accordance with the prevalent physio-psychological and anthropological views.

It explains how a relatively defective brain may stand higher as regards one special function, than a generally better developed one.

We are struck with one fact, in examining the relative development of the island of Reil in this series of animals, that if the surface area of the lobus centralis were to be arranged in a continuous series, beginning with the smaller South American monkey and terminating with man, and the area of the whole cortical expanse be arranged in a parallel manner, we should find that both series presented a remarkably similar increase. With this another observation is in accord, that where two closely related species are of different bodily size, the larger one has the more convoluted island of Reil. We see from this that the insula merely follows the general cortical development in the simian series.

A very peculiar condition is manifested by the pachyderms and ungulates: the larger species possess a distinct lobus centralis which borders, at the base of the brain, on the immense white olfactory convolution. The connection of the lobus with the basilar grey matter of the third ventricle is consequently obscured. How much of the area proper of the island is also thus obscured in some species, I am unable to determine; at all events, whatever be considered insula of such grey matter, possesses an inferior structure.

A most remarkable contrast is exhibited in regard to this lobe, by the hippopotamus and the horse. The former has a smaller cerebrum, possessing the simplest gyri, than the horse, the gyri of whose brain are as numerous and whose sulci are in places as deep as those of man! Yet the insula is small, simple and difficult of identification in the horse, extensive, prominent, and richly convoluted in the hippopotamus! We here have a repetition of the discrepancy existing between the bear and the seal, only much more intensified. In both instances the better developed brain has the worst developed insula. So that parallelism between the general cortical, and the special insular development does not hold good here, as it did in the simian and anthropoid series.

In the instance of the bear compared with the seal, I looked upon the bear's island as the normal carnivorous type, from which the seal's island was a retrogression; in the case of the two pachyderms, I consider the horse's as representing a normal type of this group, whereas the hippopotamus' manifests a hyper-development. Now the seal's atrophy found a hypothetical explanation in the atrophy of its facial and the disuse of the glossal periphery, but how can we even hypothetically explain the insular hypertrophy of the hippopotamus? The innervation of its tongue is certainly not required to be of a delicate character, nor are its facial muscles, excepting those of the nostrils, well developed. The mass of the tongue muscles and of those of mastication is immense, it is true, but this is equally the case with the muscles of the neck and the remainder of the body. For the present the lobus centralis of the unwieldy and Bœotian denizen of the Senegal, exceeding the human in its antero-posterior diameter, though only half its altitude vertically, and richly convoluted by festooned gyri in four pairs, is an enigma to the physiological anatomist.

Its upper half measured vertically is covered by the overhanging free margin of the convexity, the posterior fifth measured horizontally is covered by the mastoid lobule.* Although its gyri, on first sight, appear to run in a direction analogous to that of the other species considered in this paper, yet from their frequent alternate junctions with their fellows at the upper and lower extremities, it would seem that the influences which had determined the undulating antero-posterior course of the convolutions of the general convexity, had also had some influence on the lobus centralis, contrary to the general rule.

While the series of animals examined by the writer is far from being as complete as he could desire, yet a few conclusions can be drawn from their examination as given above.

Firstly, that the degree of development of the insula is not an indicative of the general intellectual standing of a species.

Secondly, that the innervations of the tongue and lips, which can be presumably referred to this area, or its histologically similar neighborhood in carnivores and simians, cannot be so referred in all other animal groups.

Thirdly, that even where the insula and its contiguous areas can be shown to have a relation to the function of speech, teratological deficiencies reducing it as in idiots to inferiority as compared with speechless anthropoid apes, do not incapacitate from this function. It is an open question whether the coincident defects of the cerebral organs in general in idiots may not be as important, if not more important factors in such amnesic aphasia as is present. Finally, it follows that only one element of the speech function is located in the lobus centralis and near it, that is the mechanical; the lobus is not the ruler but the mediator of this function.

PART VI.

The Olivary body in Man, the Anthropoids and lower Mammals.†

As we trace up the anterior cornu of the spinal cord into the medulla oblongata, through a series of successive transverse sections, we find that the reticular breaking up which began at the lateral cornu of Clark, now involves the anterior cornu also. This is preparatory to the disappearance of the grey matter of the anterior cornu, as a distinct mass.

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* The analogue of the human temporal lobe.

†Read before the American Neurological Association at its annual meeting, 1878.

To this breaking up there are two exceptions: the inner aspect of the anterior cornu, and that portion which corresponds to the anterior root zones, retain their masses in a concentrated form. In the altitude of the inferior pyramidal decussation the two masses are separated, higher up they join at an angle, and in the altitude of the uppermost hypoglossal nerve roots, they again separate; from this point upwards, the innermost body begins to disappear. The inner body is composed of the spongy variety of grey basis substance, and contains multipolar nerve cells of the larger kind. The lower body is composed of gelatinous basis substance, and contains smaller, round nerve cells, with few and attenuated processes.

Henle* has confounded the two under the common title of a nucleus pyramidalis, after Stilling. But since they originate separately, since their subsequent junction is accidental, and their histological structure radically different, they must be considered as two distinct bodies. The lower one has the same structure as the *nucleus dentatus olivæ*, and in certain levels is continuous with it as will be shown; the name proposed by Lenhossek, *internal accessory olivary nucleus*, ought, therefore, to be retained for it. The other division, the one containing the large multipolar cells, might retain the title of nucleus pyramidalis, if this did not lead to the erroneous conception that it was situated in the anterior pyramids, from which it is quite remote; as it lies in the inner division of the reticular formation, I propose to term it nucleus internus of the formatio reticularis. It has no relation to the olivary nuclei beyond the accidental and slight junction alluded to.

The internal accessory olive is the only representative of the olivary nuclei in the lowest mammals and in birds; it attains a high development in the parrot. It is also the first portion developed in the embryos of higher mammals, and since the proper dentated nucleus is developed from it, as will be shown,

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*Henle, *Handbuch der systematischen Anatomie des Menschen*, Vol. III., Part 2, p. 192. Figures 124, 127, 129, 132.

it is to be regarded as the fundamental olivary nucleus, although it is in simians and in man far outstripped in development by its derivative, the nucleus dentatus. Duval* states that in the cat, rabbit and other lower animals, the whole so-called olivary nucleus was homologous with the internal accessory nucleus alone, and that in these animals the external accessory olive and dentated nuclei were absent. Such a statement could be founded only on the most superficial study, and an imperfect series of specimens. All three bodies are present, side by side, in the cat, lion, bear, seal and dog; there is not the slightest difficulty in tracing the different divisions of the olive through this series. In all, the inner accessory olive is continuous at some point with the dentated nucleus, which is dentated, however, only in man and the simians; in others it is a simple U-shaped lamina.

In the seal the inner division of this U lies horizontally, and, therefore, in a straight line with the equally horizontal inner accessory olive; their continuity is interrupted here and there by the hypoglossal nerve radicles. In the bear this division of U-shaped nucleus stands vertically, and is parallel to the internal division, and the two are continuous at their highest points. In a South American monkey (Cebus apella) the lowest and innermost dentation of the main nucleus lies in a direct line with the inner body; in the baboon the latter is not a flat lamina, but a thick, roundish body, with which the inner lip of the dentated nucleus is connected as in the bear. The chimpanzee shows relations which place it far nearer the human being than to the monkeys next below it in the scale. The internal accessory nucleus here occupies a position exactly like that of man, running along the outer aspect of the pyramidal bundle. At its upper end a thin lamina runs out and downward at an acute angle, and thus continues the substance of the inner with the dentated nucleus. This same relation obtains in man, more distinctly in certain individuals than in others, and more clearly in sections through the middle part of the olive than through those striking either extremity. The continuity is interrupted here and there by the fibræ arcuatæ going from the raphe to the restiform column. Figure 129 in Henle exhibits this condition.

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* *Archives de Physiologie*, 1877.

Since the inner accessory olive is present before the dentated nucleus becomes visible in lower sections, and disappears before the latter in higher ones, it follows that only the upper half of this body is connected with the dentated nucleus.

In like manner is the external accessory olive continuous with the upper dentations of the simian and human olive, or the upper and outer half of the U-shaped olive of the bear, lion and seal. I can, therefore, consider Meynert's representation of the two accessory olives as the everted edges of the hilus of the dentated nucleus, as eminently proper and apt. It is noteworthy, that while the development of the dentated nucleus keeps pace with the general cerebral and cerebellar development, it does not reach a higher stage in the seal as compared with the bear. Schroeder Van der Kolk made an unsuccessful attempt to bring the olivary nucleus in relation to the hypoglossal nerves, as co-ordinator of the tongue, and, doubtless, the coincident deficiency of the hypoglossal nuclei and nerve roots and the relative deficiency of the olive might be utilized in a renewed examination of this theory. It is possible however that some peculiarities of the cerebellar organization, or of the restiform columns, of which the olive is an internuncial station, according to Deiters and Meynert, may lie at the basis.

The hypoglossal nerve radicles pass through the olivary body differently in different individuals and in different parts of the same medulla. They have no other relation to these bodies, and if the olivary nuclei possess any connection with the tongue, it is not with the nerve roots, but the nuclei of that organ's muscles, through fibræ arcuatæ, such a connection cannot be anatomically traced.

The Cebus has a single indentation of its olive ; the Lagothrix (a larger but related genus) has two ; the baboon has four indentations, and these separate four and five denticulations ; the anthropoid chimpanzee has fifteen, most of which are, as in man, the result of a secondary subdivision of larger folds. The largest fold is the one bordering the lateral field of the oblongata nearest the vago-accessory nerve root exits. The same fold which can be identified in man, is also the largest in the latter's case. The human olive gains additional complexity by the increase of the secondary subdivisions of larger folds. I find a series of human olives to possess, respectively, once seventeen, twice eighteen, once eighteen with an additional four upon an internal scroll-like reduplication, three times twenty-one, and once twenty-three folds, or an average of twenty. The substance of the olivary grey matter is relatively thicker in man than in the ape. but if we take into consideration the proportionate progress, shown in the series, Cebus (two), Lagothrix (three), Cynocephalus (four), chimpanzee (fifteen) and man (twenty gyri), we shall come to the conclusion that in respect to the olive as with regard to the lobus centralis, and the general convexity, the chasm between man and the anthropoid is far less than between the latter and the common Old World simians.

There are many and curious individual variations in the olivary body of man. In some cases the connecting bar with the inner accessory olive is as distinct as in lower animals; occasionally an inner scroll-like appendix is noticed, especially in richly developed brains. The most constant feature in imbecile and idiotic brains, is the compression of the outer and upper half of the olive. which is rounder and fuller in well developed human brains and flat in the chimpanzee. The outer larger fold referred to. is in such cases the one which suffers the chief reduction as to secondary folds.

In cases of pathological or teratological asymmetry of the cerebellum, I have found the olives, when asymmetrical also, which was not always the case, to follow the opposite *cerebellar* hemisphere; if the right hemisphere was atrophic. the left olive was the smaller, and vice versa. This is confirmatory of Deiters' and Meynert's statement that the olive stands in connection with the opposite cerebellar hemisphere. through the restiform decussation; on anatomical grounds alone this connection would be hypothetical.

In passing I would state, that the olivary nuclei of pachyderms present us with types quite aberrant from that just considered: that of the hippopotamus is quite bizarre in this direction, and Duval's statement may apply to these animals but I am not prepared to endorse it unreservedly, either in their case or in all the rodentia.

The external prominence of the olivary body is caused by the pressing outward of arciform fibres, through their displacement by the olivary nucleus, as has been shown by Meynert. This prominence is not always an indication of the development of the nucleus, since it is larger in the seal than in the bear, and even in individuals of our species I have found a flat eminence, due to overlapping of the anterior pyramids, to conceal a better developed olivary nucleus than another one which stood out in high relief. Few human brains can show as clearly demarcated and elevated an olivary eminence as that of one chimpanzee in my possession. In the chimpanzee whose number of olivary indentations was given, it was as prominent as in a child of the same age (two years).

The eminence is very distinct in the baboon, bear and seal, and although much narrower in the feline and canine carnivores, is yet unmistakably present, although Meynert* denies this. Doubtless his specimens had been permitted to rest on their basal face too long, obliterating their demarcating depressions.

I have not specified in this paper that my description deals only with that olive which is termed "inferior" by writers, in contradistinction to a body in the trapezium described as the "superior olive." I think that the necessity of making special mention of the topography of these bodies can be obviated, by terming the lower body the olive *par excellence*, and the upper, the "nucleus of *the trapezium*," especially as the so-called superior olive of Luys is an entirely different body from that described by Meynert, Henle, Dean, Clarke and others under that title.

PART VII.

The homology of the corpora quadrigemina, and some observations on the posterior longitudinal fasciculus.

Since the comparative development of the nervous system has occupied the attention of anatomists, it has been regarded as a fundamental difference between the mammalia and sauropsida, that only the former possessed corpora quadrigemina, their place in the latter being occupied by bigeminate optic lobes. Neither proposition, although adopted in zoological classification, is accurately correct.

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* "Die Gehirnbau der Saugethiere." *Stricker's Handbuch der Gewebelehre*, II.

The first steps toward correcting this mis-statement, were made when it was shown that only the anterior pair of the corpora quadrigemina were connected with the optic tracts. Since the entire optic lobes of those reptiles possessing only a single pair are connected with these tracts, it follows that, physiologically, at least, only the anterior pair of mammals is represented in the optic lobes of birds and reptiles. Forel makes this deduction, and suggests that in the singular monotreme ornithorynchus, a transition between the mammalian and reptilian form of the optic lobes may be found. I am able to present a different solution of the problem.

In examining the brains of two large snakes of the anaconda family, I found distinct corpora quadrigemina. The posterior pair is as clearly demarcated from the anterior by a transverse sulcus, as the members of the anterior and posterior pairs are separated from each other by the longitudinal sulcus. The posterior .pair is distinctly tuberculate, and measures about one-third the dimensions of the anterior.

In a series of transverse sections made through one of these brains, I find that as in mammals, the optic tract enters only the anterior pair.

A single instance like this, destroys the validity of the characterization of the sauropsida as a class possessing only two lobes in the mesencephalon, and I made a renewed examination of the conditions obtaining in other reptiles. In several families, but notably well developed in the turtles, I succeeded in discovering the homologue of the posterior pair, as a concealed ganglionic mass, lens-shaped, and containing the same elements as the posterior pair of the anaconda. It is concealed under an arciform fibre-mass continuous with the cerebellar medullary lamina, and lies directly behind the optic lobes properly so called. It has no connection with the optic tract, and appears to be in relation with the sensory root of the fifth pair.

From this it follows, that reptiles as well as mammals possess the homologue of the posterior pair of the corpora quadrigemina; that while in most reptiles this homologue is separated from the anterior pair, and concealed under other structures, it arises to the same topographical level in the python family as in mammals, and finally that the posterior pair and its homologues are in no respect ganglia of the optic nerves. I can, therefore, fully assent to Forel's statement that the two pairs of the corpora quadrigemina are neither subdivisions of one common ganglion, nor even analogous ganglia, such as Meynert would make them.

As a rule in the animal range, it seems that the higher a zoological family, the more distinctly do the posterior tubercles become demarcated and developed. The minimum is in the anaconda, and from this point, of the animals examined, the opossum, rabbit, dog and man, represent an ascending series. These bodies are very small in the opossum. In comparing the relative development of the anterior and posterior tubercles, those animals only should be placed in the series which have well developed eyes, for a defective visual apparatus, associated as it always is with rudimentary anterior tubercles, leads to an apparent but deceptive absolute preponderance of the posterior pair, which in reality is only apparent, as in the bat and mole.

In view of the entirely distinct character of the anterior pair of the corpora quadrigemina from their posterior pair, it would be desirable to re-examine the embryonic development of these bodies. His, in his lectures (1874), stated that the single mesencephalon was first divided into two lateral halves by a longitudinal sulcus, which was subsequently crossed by a transverse sulcus, dividing it into four divisions. I doubt whether this statement was based on a sufficiently extensive series of observations made on mammals.

In reptiles the optic lobes exhibit a true cortical structure, and the cortical lamina is covered on both sides by white substance. The outer consists of fibres entering the optic tracts, the inner of projection fibres of the second category, which run parallel with the contour of the Sylvian aqueduct, and in part decussate beneath its floor.

This is the homologue of Forel's fountain-like tegmental decussation.* The innermost fibres of this mass pass into the oculo-motor nucleus of the same and of the opposite side, still others which fail to decussate, leave their original direction and become longitudinal, constituting the posterior longitudinal fasciculus.

This fasciculus, thus shown to originate in the deep white substance of the optic lobes, probably has the same origin in mammals, since in no animal can it be traced beyond the posterior commissure. Forel has already supposed such an origin for it. I am very much inclined to doubt whether any statements that this fasciculus originates as Meynert, Huguenin and Rohon† claim, in the cerebrum, or the sublenticular grey masses, are based on the comparison of a sufficient number of sections.

Flechsig and Forel consider this fasciculus as a connecting bond of the oculo-motor nuclei and the spinal grey substance. It can certainly be traced with great clearness into the column of Tuerck, especially in lower animals. In a series of longitudinal sections of the cat's brain, I find that it sends a distinct branch into the trochlearis nucleus, in which it becomes lost. The abducens nucleus is entered by a similar bundle, whose connection with the longitudinal fasciculus cannot be traced; it certainly does not come from the raphe, and I consider it derivable from the former by exclusion.

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* Forel, "Untersuchung ueber die Haubenregion und ihre oberen Verknupfungen, etc." *Archiv fuer Psychiatrie*, VII.

† Rohon, "Das Central Organ des Nervensystems der Selachier." *Denkschriften d. Kais. Acad. der Wiss.*, 1877.

(*To be continued.*)

ART. III.—NERVOUS EXHAUSTION OR NEURASTHENIA IN ITS BODILY AND MENTAL RELATIONS.

ONE OF A COURSE OF LECTURES ON THIS SUBJECT, DELIVERED AT THE CHICAGO MEDICAL COLLEGE, BY J. S. JEWELL, M. D., SESSION OF 1878-9.

LECTURE I.

ANALYSIS.—Definition—General prevalence—Not a new disease—Names which have been applied to it—Two classes of Phenomena, bodily and mental—*Motor Phenomena*, loss of power and augmented reflex excitability—Striped and unstriped Muscle—Vaso-motor reflex, exalted—Effects of same, direct and indirect—Heart—Small vessels—Secreto-motor nervous system—Its reflex excitability increased—Loss of will power or control—of capacity to do or endure in the sphere of mental action—*Sensory Phenomena*, sensibility of all kinds exalted—Hyperæsthesias of general and special sense—Undue emotional excitability—Melancholia—Summary.

GENTLEMEN: The subject of the next few lectures, is one of great practical importance. It is *Nervous Exhaustion*, or *Neurasthenia*. This latter term seems not inaptly chosen, and means, of course, lack of nerve-force, or nervous exhaustion. It does not mean, however, the temporary exhaustion produced in a healthy, strong person, suddenly, by fatiguing effort, and which may be repaired by a full meal and a night's sleep. It is rather the comparatively permanent exhaustion which is the result of prolonged over-strain, mental or physical or both, too little rest, insufficient or defective nourishment, long continued, until the substance of the nervous system, and often of the blood which nourishes it, is wasted or worn away, far below healthy limits, entailing as a necessary consequence, a corresponding *loss of nerve power*, and in most cases *morbid exaltation of nervous sensibility*, not to speak at present of other important, though auxiliary elements of such cases. The two factors I have placed in the foreground are the essential ones in every case of Neurasthenia. They are, as a rule both present, loss or lack of power always is so. Neurasthenia may be exhibited in the sphere of the mind as in weakness of thought, but especially weakness and vacillation of will, or in other words, loss of control and lack of decision; in unduly excitable and unhealthy emotion, generally of a distressing or depressing character; or in the sphere of the simple physiological activities, especially in feebleness and irregularity of muscular action; exaltation of physiological sensibilities, of any or all kinds, but especially *reflex* excitability; the circulation of the blood is also, as a rule, unsteady and fluctuating, the action of the heart and small muscular vessels being very easily disturbed by multitudinous causes, mental and physical. Then again it may be general, extending to the whole nervous system, or it may be strictly localized, involving only limited parts of the nervous system, while other parts do not participate to the same extent or at all. Again it may be hereditary, or congenital, especially the former. People are being born into the world daily, to begin, live, and end life, neurasthenic, or at least to be brought easily into that state; persons who have from the beginning weak, tricky nervous systems, either as a whole or in some of its parts. On the other hand, there are persons to be met with everywhere these times, at all periods in life, and of both sexes, who are naturally healthy, as this phrase goes, who from prolonged over-exertion, mental or physical, or from being under a load of care or distress, or from prolonged loss of sleep, or loss of appetite, or from insufficient food, or on the contrary, excessive waste, as in hemorrhages or diarrhœas, or on account of some irritating, wasting disease, etc., suffer a loss of balance in their nutrition, so that waste for a long time preponderates over repair, to the exhaustion of the nervous energies, and a great gain in mobility, or "shakiness" of the nervous system, especially the nervous centres.

It may form a disease (as it must be called) by itself, or it may and it does form a more or less conspicuous element in a great variety of diseased states, one which it behooves you all to learn to detect and estimate. It is often overlooked or under-estimated, and often is the obscure and all-important element in cases which are widely different in appearance. It is the undertone in the picture in a vast number of cases of "heart disease," "brain disease," even "softening" of the brain, of hysteria, epilepsy, melancholia, neuralgias, paresis, mental weakness, feeble circulation, insomnia, &c., &c. It prevails at all periods in life, and in both sexes. Instead of becoming less common, it is becoming more so, as time passes, and as people as a whole become more sedentary in habits, more intellectual in activities, more engaged by occupation or by culture, so as to augment the sensibilities at the expense of the forces or power of the nervous system.

No physician can practice medicine and not meet with it, and the minority is a small one, in the profession as a whole, to recognize it clearly, and comprehend its underlying conditions, as it seems to me they may be.

It is not a new form of disease. It has been long recognized, and has had widely different names applied to it. And yet, as already said, but few recognize it.

It has been extensively written upon by certain French authors under the name of *nervosisme*, especially by Bouchut. It has been treated of by MM. Sandras and Bourgignon, under the name of the *nervous state* (l. etat nerveux), by Cerise, as *nervopathie proteiforme*, as *nervospasmie* by Brachet, &c. Among German writers it has been recognized, at least in some of its forms (to go no farther back), since the time of P. Franck, as *spinal irritation* (Stilling and others), as *Nerven-erethismus* (Henle, *Rationellen Pathologie*), *Nervosismus*, &c., by others. In England it has been described since at least the time of Robert Whytt, under various names, such as spinal irritation, nervous exhaustion, neurasthenia, neuræmia (Laycock), cerebrasthenia, &c. In our own country, it has been partly discussed by various writers, among them being Drs. Hammond, Mitchell, Beard, and others. But it is no part of my present purpose to trace the history of the literature of the subject. I have wished merely to inform you that it has been long recognized and often written upon, and hence is not a new form of disease. It is my purpose rather to give you the *results* of my own studies, not only into the writings of others, but much more of my own practical observation and experience of the disease, if it may be called so.

In what I may have to say, I will describe it with some particularity in its more important relations. In doing this I will first speak to you of its signs or symptoms. In the next place I will try to analyze the phenomena so as to get at their most simple classification and expression. This much done, I will endeavor to show what are the organic conditions which may be supposed to underly the symptoms. Finally, I will endeavor to point out to you the causes and treatment of the, at first sight, complex phenomena which may be grouped under the head, *Neurasthenia.*

In the endeavor to describe the visible or palpable phenomena of neurasthenia, it may be first noticed, that they fall into two great groups, *bodily* (somatic) and *mental.* The same organic conditions underlie both classes of phenomena, the differences being in the parts of the nervous system in which the morbid states exist. Having distinguished two classes or groups of phenomena belonging to nervous exhaustion, I will call your attention to the class of bodily symptoms. This distinction of the one group from the other, it must be remembered, is for practical purposes only. I would not have you for a moment suppose the distinction is given as a radical one, whatever may be the truth as to a substantial difference between mind and body. To attempt to decide that question is outside of my present purpose. The bodily symptoms or signs of neurasthenia fall chiefly into two groups, *motor* and *sensory*, for in the sense I now employ the term, neurasthenia is *a diseased condition of the nervous system par excellence.* Belonging partly to the motor and partly to the sensory spheres, but worthy on some accounts of being considered separately from either, *is an easily disordered circulation.*

1. *Motor phenomena.* These consist always in loss of muscular power and endurance, and as a rule comprehending exalted reflex excitability toward muscular action. The patient shows a permanent lack of muscular power, its volume, so to speak, being less than it is in the same person in health. This may depend, of course, to some extent on the condition of the muscle itself. If from disease, or from over-use, or from defective or insufficient nourishment the volume of a muscle

is diminished or its structural integrity impaired, no matter what may be the condition of the nervous system back of it, the power of the muscle will be under par. It is necessary to take this circumstance into the account in the study of neurasthenias, as indicated by muscular weakness, for this latter is not always a measure of the former. The same may be said in a less degree, perhaps, of *endurance* in muscular action—or the ability to continue in exercise for a certain length of time without marked fatigue or exhaustion. In some cases an individual, even in apparent health, and with every appearance of a normal volume of power, is speedily exhausted in the progress of moderate muscular exercise. The stock of energy which appears as if ample to begin with, is soon exhausted, and the individual is said to lack "bottom" or endurance. In such a case there is a real neurasthenia, apparent at last, though it may not be so at first.

The distinction between the volume of nerve power which may be available for immediate effort, and endurance, irrespective of the *latter* point, is one of some importance. For it often happens they bear no direct proportion to each other. At times a person is met with having remarkable endurance or tenacity, and a very small volume of nervous and muscular force available for immediate use, frail, weak, exhausted as permanent conditions. Such persons will often pass an ordeal in which others quite robust in appearance would perish. These points may be especially useful for you to remember and accurately estimate in making prognoses, and in laying down courses of treatment, especially in the matter of hygiene.

Then, as already said, there is *exalted reflex excitability.* This is present as a rule in all cases of nervous exhaustion of the fixed kind I am considering in these lectures, and in many cases its augmentation is remarkable, as in hysteria and chorea, which, as a rule, have in them as a major factor, neurasthenia, as I shall soon try to show you.

You will, of course, all recall what reflex action is, and its incalculable importance in the physiology of the nervous system, especially in its relations to muscular action. I will assume that you are acquainted with its facts and laws. But I may remind you of the fact that it implies in its simplest expression, two nerve fibres and a nerve cell—one fibre from a peripheral region leading into the cell, and another out of the cell, to some part, say a muscle, the action of which is capable of being affected by an impulse from the cell, emitted in answer to an impulse conveyed into it by the ingoing fibre. Now if the impulse going out from the cell to the muscle is stronger or more easily aroused than it should naturally be, one or both of two things may be true: Either the ingoing fibre is more strongly excited than it should be, or the nerve cell is for some reason more responsive than it should be. Both of these circumstances may be present, of course, but usually only one, perhaps. But whatever may be said as to the strength of the peripheral excitation, in any case of neurasthenia in which reflex excitability is exalted, it must be said that the condition of the cell, as regards its nutrition, is one which makes it more responsive than when normal. As the cell becomes leaner and less firm in texture, it becomes more excitable than it should be when more solid and perfect in its intimate structure. Under such circumstances very slight causes will provoke reflex muscular action. A sudden or unusual sound, impressions made on the eye, impressions of various kinds made on the skin, excitation of the alimentary mucous membrane, or of the genito-urinary or other sensitive parts of the body; and besides these, excitations from the cortex of the brain descending the fibres of the corona radiata, and other bundles of fibres in the interior of the brain, to the apparatuses of cells of the basal system of ganglia, of the medulla and spinal cord, often beget reflex action, which, when it occurs, is apt to be wide-spreading, and is said to be due to emotional or mental disturbance.

From all these sources the excitations may come to provoke reflex muscular action, and the readiness with which it is excited depends on the condition of the nutrition of the nervous system, more particularly the centres, a condition which certainly obtains in neurasthenia.

But reflex action is not alone manifested in the voluntary muscles, but in other and even more important ways. In no other way is it shown in a more striking manner than in a disturbed circulation. As you are now aware, the heart sustains not only very important relations to the medulla and spinal cord, through the medium of the vagus, accelerator, depressor and vaso-motor nerves, but it has an automatic, and as we may say, *intrinsic* nervous apparatus of its own. Then again, you will remember that the small muscular blood-vessels, especially the arteries, are all of them provided in all probability with peripheral nervous apparatuses, sown broadcast throughout the body, upon which the vessels depend in a most important sense for their *tonus*, or tightness of embrace of the column of blood passing through them. These multitudinous little nervous apparatuses act as do other parts of the nervous system, in a reflex way, and hence they are liable to become more irritable and responsive to impressions of one kind or another, as well as other parts of the nervous system. But farther, these peripheral vaso-motor mechanisms are connected with the grey matter of the spinal cord and medulla by nerve fibres, perhaps by two kinds of fibres, one of them so acting on the peripheral apparatus as to increase, and the other so as to diminish its activity. The former pass under the name of vaso-constrictors, the latter under that of vaso-dilators. The central vaso-motor apparatuses of the cord and medulla, and the nerves just described as proceeding outward from them, may be, and are affected with remarkable ease and frequency in a reflex way. In this manner the action of the heart may be disturbed so that palpitations, variations in the force and frequency of the pulse, &c., may and do occur. Also variations in the tonus, or condition of contraction of the small muscular blood-vessels may occur, either throughout the whole body or in some limited portion of the same, all by reflex nervous action. These varying conditions of the circulation depending on the condition of the vaso-motor nervous system, and if extreme in extent or in degree, may give rise to a vast variety of symptoms depending on the seat, or degree, or duration of the circulatory disorder. This kind of disorder is almost characteristic of neurasthenia.

Vaso-motor reflex excitability is in general conspicuously exalted. Comparatively slight impressions, whether physical or mental, may profoundly modify, in a reflex way, the action of the vascular system, and through it all the functions of the body, since all depends, in a certain sense, on a due distribution of the blood. A word, an unusual or a sudden sound, a strong light, impressions of cold and heat on the skin, or of other kinds, irritation of the alimentary tract, from the mouth downwards, irritation of the genito-urinary apparatus of all kinds and from all causes, all emotional states, if violent or sudden in occurrence, all muscular or mental effort if violent or long continued, all exciting agents of a medicinal kind, &c., &c., may affect the action of the vaso-motor nervous system, and through it the action of the heart and muscular vessels, and so on to the end of the chain of evil consequences which follow, and act, and re-act on each other often in a surprising manner. In this case they can be chiefly traced back to a worn, exhausted, and hence unstable, over-sensitive, vaso-motor nervous system.

The symptoms which grow out of the fluctuating circulation, as just described, are legion. I could not pretend to enumerate them fully. But, if you will, once for all, get clearly in your minds the notions I have advanced in regard to reflex vaso-motor action, and its exaltation in neurasthenia, I feel pretty sure you will soon learn to recognize and construe them correctly.

Then again, the *secretions* are as really under the control of the nervous system as the muscular tissue is of the blood-vessels. There are secretory nerves, if not a secretory nervous system. The researches of Bernard, Kuehne, Prevost, Adamkiewicz, Luchsinger, Elischer, Nawrocki, Vulpian, and many others, have established beyond question, so it seems to me, the existence of a *secreto-motor* nervous system, not only a central spinal and medullary apparatus, but also of nerves which go out from the same to the various secreting organs of the body, and the actions of which are, in an important sense, under the control of these nerves. But I cannot at present go into this subject, only so far as will enable me to show you that the secreto-motor nervous system may be unduly sensitive, and may be affected in a reflex way as certainly as the musculo-motor nervous system can be. As examples, I would refer to the notable variations of the urine in quantity which occurs in persons when in a neurasthenic state. This is especially so in cases of neurasthenia in nervous females, in which there are certain symptoms growing largely out of the neurasthenic state, called hysterical. I would also call your attention to the profuse " nervous sweats," which are often seen in neurasthenic persons under irritation or excitement. Besides these, other secretions are affected, either in the way of increase or decrease, under such circumstances that we seem obliged to refer them to increased mobility of the nervous system, especially to increased reflex excitability of the nervous system, which certainly is a prominent element in neurasthenia.

Then there is, generally speaking, increased sensibility, whether *general* or *special*.

The skin is as a rule hypersensitive as a whole or in places. In some extreme cases a touch on almost any part of the body is painful, or, at least, disagreeable, a slight touch being often more painful than firm pressure. The increase in cutaneous sensibility may relate only to tact or touch, but is more likely to relate also to the pain sense and temperature sense.

Sudden changes in temperature, especially from warm to cold, produce painful shocks or impressions, far more than in health in the same individual. Very slight injuries to the surface are comparatively more difficult to be borne than in a healthy condition of the nervous system. It is more easily disturbed or shocked, because it is less solid and resisting than in health. Cutaneous sensory impressions arouse reflexes more easily and of a wider range than we see in health under the same circumstances. This partly arises from the loss of *inhibitory* power of the nervous system, or of that control of the higher nervous centres over the reflex actions of lower centres. While the reflex excitability of these latter is exalted, as I have said already, the power of restraint, control, guidance, which higher centres exert over lower ones, is weakened. This form of nervous force is diminished in neurasthenia as well as other forms of nerve force. All these various forms of nervous force are at bottom the same. The difference is not in nature, but in modes of manifestation. *Loss of control* or of will power is, indeed, one rather clear mark of neurasthenia. There is also as a rule undue sensitiveness of the special senses. Photophobia, more frequently asthenopia and undue, even painful acuteness of hearing, is very generally present. Smell and taste, especially the former, are often morbidly acute, but not in so striking and disagreeable a manner, nor with the same constancy as the other forms of sensibility mentioned.

As regards mental action, there are several common features worthy of particular notice. One of the most striking of these is loss of power, especially will power, and the ability for prolonged or severe effort. Such a person can as a rule neither do nor endure what was possible when in health. Cares sit heavily, and anything requiring much thought or calculation, and hence involving the expenditure of much brain force, becomes difficult or practically impossible. The memory is slow or defective as compared with what is natural to the individual. Then again, another feature is excessive emotional excitability, especially in a painful or depressing way. Such persons are comparatively too easily affected. Slight circumstances annoy or distress them, or excite them. They are especially apt to take a depressing view of their business affairs or of their own health. They feel themselves often unable for the duties of life, and may and do readily fancy themselves the subjects of different serious or fatal diseases. They have very frequently the "blues," or mental depression, all the way from a slight temporary despondency to the most persistent and profound melancholia, which may assume every possible form of this distressing disorder. The neurasthenic patient cannot sleep well, in the majority of cases is easily wakened, and when sleeping, not uncommonly has dreams at times quite unpleasant.

Such, gentlemen, are the principal morbid phenomena which are manifested in neurasthenia. It is not an exhaustive description of them. It is only a general outline derived from a comprehensive analysis of such cases. To undertake to describe the various kinds of cases with any kind of fullness would be an impracticable task. But it is possible by analysis and classification for us to describe the principal phenomena of this protean and complex affection. They may be summarized as follows :

I.—SOMATIC OR BODILY PHENOMENA.

1. Exalted physiological sensibility, general and special, including reflex excitability.

2. Permanent lack of nervous power, including power of inhibition.

3. Beneath all this, impaired nutrition, in which there is a more or less permanent preponderance of waste over repair, especially in the nervous structures of the body.

II.—MENTAL PHENOMENA.

1. Exalted emotional susceptibility and mobility, especially in the direction of depression.

2. Loss of will power or power of control, and in general not of mental *sensibility*, but of mental *power*, to do or endure within the sphere of mental life and action.

3. Underlying all of which are combined nutritional and circulatory lesions, especially of the brain, which impair the substance, diminish and render unsteady the exercise of the power of the brain, and finally exalting its sensibility.

Under one or other of these heads, may all or most all the phenomena of neurasthenia, or nervous exhaustion, be ranged.

Now let these statements be kept in remembrance, and they may serve to guide us in the next lecture when I shall try to show you what are the organic conditions underlying these and similar symptoms, and morbid conditions.*

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* There will be two or three more of these lectures, all of which were given during the course of instruction in Nervous and Mental Disease in the Chicago Medical College, for the Session of 1878-9.

Art. IV.—A CASE OF PROGRESSIVE MUSCULAR ATROPHY WITH SCLEROSIS OF THE LATERAL COLUMNS.

By Dr. J. C. Shaw,
President New York Neurological Society, Medical Director Kings County Insane Asylum.

THE following case is of interest in its relation to the pathology of progressive muscular atrophy, and also in view of the recent division made by Prof. Charcot of this disease into two groups. I have to thank my friend Dr. B. A. Segur for the opportunity of seeing this patient. The first part of the case was observed by him, the latter part by me.

Male, aged 44, with apparently no neurotic family history; at 20 years of age he had gonorrhœa, followed by stricture, gleet and its accompanying pain in back, hips, &c., but otherwise he enjoyed good health. In 1870, his first impairment of health began; in February of this year, after an exposure to wet and cold, he became quite sick, the exact nature of which sickness is not known; it was of such a nature, however, as to confine him to his room for two and a half months, and he was unable to engage in any occupation for one year after; he thinks he never entirely recovered his former vigor; following this he had some pulmonary hemorrhage and expectoration, which was slight. In October, 1875, the first symptoms of his present difficulty appeared; he began to notice fibrillary twitchings in the small muscles of hands, especially those of the thumb, and simultaneously there was a wasting of these muscles, their function was also impaired, as shown by inability to tear up pieces of paper, &c., which is done by the index and thumb; the condition was alike in both hands. The atrophy went on so rapidly in the muscles of the hands that in January, 1876, he was unable to write; the atrophy and fibrillary twitchings then began in the muscles of the arm and forearm. He began to have bulbar symptoms about May of this year, and by October the glosso-laryngeal paralysis was quite marked, and his speech was almost unintelligible; at this time he was also unable to walk with his accustomed firmness, tiring easily.

I first observed him in May, 1877, when his condition was as follows: Unable to utter a word, could make simply a grunt, tongue immovable in floor of buccal cavity, a mass of furrows and wrinkles, and the seat of continuous fibrillary contractions; he has the remains of a harelip, a good deal of difficulty in swallowing, upper extremities and chest a skeleton, the muscular atrophy has become so great arms hang by his side quite flaccid and limp, no contracture present, fibrillary contractions wherever there is any muscular tissue left, lower extremities in good condition to inspection, patient can stand and walk but is easily fatigued, no anæsthesia, no rectal or vesical difficulty, the atrophied muscles are the seat of soreness, and when the muscles are pressed or stretched it causes discomfort and pain; what muscular tissue is left reacts normally to faradism; this condition advanced, and the lower extremities began to atrophy rapidly, and in the middle of August, 1877, he was unable to stand, and had to keep his bed; he existed in the most deplorable and helpless condition until September 27, 1877, when he died, evidently from exhaustion and the bulbar disease.

Post-mortem twelve hours after death. Unfortunately I had to make the examination in a great hurry. The spinal cord and the brain were the only parts examined by me. I regret not having been able to examine the peripheral nerves and the muscles. The pectoral muscle was the only one examined; to the naked eye it had a pale appearance, what was left of it; the cerebrum to the naked eye presented nothing abnormal; the spinal cord also showed nothing unusual when intact, but when transverse sections were made in it at various points previous to hardening, the grey matter was seen to be much more vascular than normal; the anterior nerves were apparently not diminished in size, but presented an unusual translucent appearance.

Histology. Specimens hardened in solution bichromate potassa; the cord was divided into fifteen segments of about equal length, and transverse sections made from each of these segments; stained in carmine and extractum hæmatoxyli and mounted in Canada balsam after Lockhart Clark's method.

The cervical region was the point at which the disease began, and was examined first; here the morbid changes were of a very marked character; examination of the sections made from other regions (of the cord) soon showed that the changes which had taken place were identical throughout the cord, but differing in degree and in some minor respects, but differing in such a manner as to render important aid in the interpretation of some of the points in the pathological physiology of the case, and in the sequence of the pathological changes which are seen to be present. Two very decided pathologic changes are seen in the cord:

1st. Atrophy of the ganglion cells in the anterior horn.

2d. Sclerosis of the lateral columns.

The atrophy of the ganglion cells of the anterior horn in the cervical enlargement is very nearly complete. In almost every section nothing is to be seen but a very small mass of yellowish brown pigment, here and there, occupying the place of the cell. In only one section from this region did I find a cell in fair preservation; but without any processes, and containing a considerable amount of pigment, but having a nucleus and a fair amount of protoplasm around it sharply stained by the carmine.

In the dorsal region the cells are almost as much atrophied, but as you pass down to the lower dorsal region you find more and more cells in a fair degree of preservation; none, however, in a normal condition. In the lumbar region, the largest amount of cells are to be seen, and in some places the number is almost normal, but there is not one cell which has not been affected; the pigmentation, atrophy and the absence of processes are the characteristic changes in these cells; the pigmentation takes place in the body of the cell, usually at one edge, and encroaches upon the nucleus; the pigment is of a yellowish brown color; the atrophy of the processes appears to come about in an entirely different way, they become more and more translucent and attenuated, and at last melt away, from the distal end first, without any more positive change, and they refuse to take up the carmine; this method of disappearance of the processes gives a peculiar appearance to the stump; it looks as if the process was cut off somewhat abruptly, not sufficiently so, however, but as to leave behind a vestige of the process, showing this peculiar translucent appearance, which, in some cells, contrasts very decidedly with the thick, sharply stained body of the cell; it would appear that all the processes do not undergo this mode of change, for occasionally there is to be seen in the grey matter of the anterior horn a few processes in fair condition, which evidently have been cut loose by the section knife; these, however, are very few.

The sclerosis of the lateral columns is most intense in the cervical enlargement, and becomes less so as we go down to the end of the cord; the accompanying wood-cuts show the distribution of the sclerosed area; it differs greatly in the different regions; the sclerosis is of a very light character throughout the cord; it is a very limited region in which the nerve tubes are much altered; the nerve tubes in the sclerosed region have simply become a little smaller, have a cloudy appearance and a tendency to run together, and a light granular material around them; the septa running between the nerve bundles are a trifle thicker, and, therefore, appear more numerous in this region, and take up the carmine staining very sharply, so that to the naked eye the sclerosed region appears more diseased than microscopic examination shows it to be in those sections; from the cervical enlargement quite a number of spider cells are to be seen, similar to those described by several authors in various pathological conditions of the nervous system, also by Dr. E. C. Seguin and myself in disseminated cerebro-spinal sclerosis;* but they are quite small; in the other regions there appears to be very little change in the neuroglia cells; there is an increase of blood-vessels in the diseased regions, grey as well as white matter; everywhere the blood-vessels appear normal. In the cervical region the central arteries are surrounded by, and their perivascular spaces distended with an opaque material which is lightly stained by hæmatoxylin, and which I take to be an exudated fluid of some kind, albuminous probably, and which has become coagulated; moreover, there is a cloudy appearance in the entire diseased area in this region, grey as well as white matter, and which is evidently due to an infiltration of the same kind of material; the question of its being a defect in the method of preparation was soon settled by making many sections in different ways, and the additional facts that in the same section those parts of the cord remaining normal were not in the least cloudy, and that the dorsal and lumbar regions prepared in the same way, did not present the same appearance. Sections made very near together, through the decussation and into the medulla, showed that the sclerosis extended into the anterior pyramids of the medulla; but the sclerosis became more and more light until, in sections made through the middle of the olivary bodies, it was a very difficult matter to tell if there was any sclerosis present; but after making a great number of sections through the decussation so as to find where the sclerosis ceased, and, after a most careful examination of all the specimens in their order, I was able to satisfy myself that there is a very slight sclerosis here, and that it has been growing less and less intense up to this region; but I was also able to determine that there is a slight sclerosis in this region (sections through middle of olivary bodies) in that part of the medulla which Meynert considers as belonging to the lateral columns of the cord (see Fig. 4, B), and that a very light fine strip extended from this down almost to the pyramids (see Fig. 4). Almost all the nerve cells in the nuclei of origin of nerves have suffered atrophy, the cells in the pneumogastric nucleus are wasted and indistinct, but the nucleus of the hypoglossus is where the disease is greatest, the cells have undergone extensive pigmentation and atrophy, not one nerve cell is to be seen with a process; the cells in the nucleus of the sixth and seventh nerves are in good condition.

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*"A Contribution to the Pathological Anatomy of Disseminated Cerebro-Spinal Sclerosis," Drs. E. C. Seguin, J. C. Shaw, and A. Van Derveer, JOURNAL OF NERVOUS AND MENTAL DISEASE, April, 1878.

Sections through the middle of pons and crura (left) shows nothing abnormal.

The pectoral muscle shows simple diminution in the size of the muscular fibrils, and nothing more.

The question immediately arises, what is the connection between the symptoms in this case and the pathologic changes found? Numerous cases have now been reported in support of the spinal origin of progressive muscular atrophy, and this case is an addition; this view we can now consider almost definitely settled; the myelitis of the anterior horn and disappearance of the ganglion cells explains the muscular atrophy; but as to the connection which the lateral sclerosis bears to the myelitis of the anterior horns and to the symptomatology we are still in doubt.

In recent years Prof. Charcot* has announced and taught that what has heretofore been called progressive muscular atrophy really comprehends two distinct diseases, having a special symptomatology and distinct pathological anatomy; a first form he calls progressive muscular atrophy protopathic (type Duchenne-Aran), a second form progressive muscular atrophy, deuteropathic or lateral amyotrophic sclerosis.

In the first form muscular atrophy is the first and predominant symptom, the disease lasts many years, and bulbar symptoms do not occur; myelitis of the anterior horns is the pathological condition; in the second form, first paralysis or paresis, then muscular atrophy and contracture, finally bulbar paralysis; the disease lasting about three years, with fatal termination; pathology, lateral sclerosis and secondary myelitis of the anterior horns.

Now what is my case, one of lateral amyotrophic sclerosis with bulbar paralysis?

Clinically, the contracture which Charcot appears to consider as constant is wanting, there is not even the least contracture in the hands, and the muscular atrophy here was certainly the first symptom; the rapidity of the case and the bulbar paralysis corresponds to the clinical picture given by Charcot of lateral amyotrophic sclerosis. Now, when we come to examine the cord there are what appear to be at first sight changes which place the case as one of lateral amyotrophic sclerosis, but a most careful study of the sections lead me to consder it otherwise.

These are my conclusions: the myelitis of the anterior horns (commencing in the cervical enlargement) was the primary lesion; the extent and severity of the lesion and the time of

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***Archives de Physiologie*, 1875, and *Maladies du Systeme Nerveux*, Tome II.

appearance of the muscular atrophy lead me to this opinion; the lesion in the lower part of the cord and in the analogues of the anterior horns in the medulla was simply a matter of extension; the sclerosis of the lateral columns I look upon as entirely secondary, and rather as a form of secondary degeneration than a true sclerosis; I cannot conceive how a sclerosis of the lateral columns can exist for such a length of time (if it had been the primary lesion) and yet produce so slight an effect on the nerve tubes as was found to be the case here.

I have spoken of this condition of the lateral columns as sclerosis, because it has been so called by most observers, but to me this appears incorrect, for there certainly is a difference between this change and that which takes place in disseminated cerebro-spinal sclerosis, and to this latter condition would I restrict the word sclerosis as indicating peculiar, active, and primary changes in the neuroglia cells; the condition spoken of in this case is, as far as the neuroglia cells are concerned, comparatively a passive one, and resembles more the condition seen in so-called secondary degeneration due to primary brain lesion, where the nerve tubes appear to be the primary seat of trouble; the same appearance of the neuroglia cells is seen in the sclerosis of the posterior columns in locomotor ataxia, and is a different process from that in disseminated sclerosis.

The presence of contracture in these cases, as a symptom, which is always present and due to the sclerosis of the lateral columns, and which is one of the distinguishing points of this variety, as Charcot supposes, is certainly not correct. I have had the opportunity of seeing another case similar to this one which I describe (but in which I obtained no post-mortem). Here there was no contracture, and Prof. Leyden has recently recorded several cases* in which post-mortem showed, like my case, very light sclerosis of the lateral columns; contracture was absent in several of his cases. Leyden's cases, although beginning with bulbar symptoms, are evidently the same disease, having its origin in a different point of the cerebro-spinal axis.

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* "Ueber progressive, amyotrophische Bulbarparalyse, etc." *Archiv fuer Psychiatrie und Nervenkrankheiten*, Band 8, heft 3.

The connection between the lesion in the grey matter and that in the lateral columns is one which has created much interest and some discussion. Charcot is the father of the view that in those cases which present the clinical history and symptomatology, as previously given, and in which microscopic examination has shown myelitis of the anterior horns and symmetrical lateral sclerosis, that the sclerosis is primary and the lesion of the anterior horn secondary. Upon this he has founded the disease, lateral amyotrophic sclerosis. This view of Charcot's has been pretty widely accepted, and recently Gombault* has published a monograph on the subject; it has, however, met with sharp criticism from many quarters, especially German neuropathologists. At this time it appears impossible to come to any very definite conclusion on the subject. Many more observations will have to be recorded before it is settled; this, however, is certain, that there are lesions of the grey anterior horns which will produce secondary lesions of the lateral columns. The idea that the lesion of the lateral columns is due to a bilateral lesion in the cerebrum is not confirmed, and appears, to say the least, rather doubtful. It is true, as Flechsig says, that the lesion is most intense in what he calls "Pyramidenbahnen" (at least it is so in my case), but it certainly is not equally intense throughout this bundle, but rather occupies the inner part of it; and the idea that the lesion shades off from this is to me incomprehensible, as the shading off, if it were so, goes almost entirely to the anterior white matter. The sclerosis being most intense in the cervical region, shows that it is not dependent upon lesion above the cord, and the fact that the sclerosis disappears in the medulla, supports this, and argues that it is dependent upon the myelitis of the anterior horn. I do not deny that there is a primary lateral sclerosis with secondary lesion of the anterior horns; but there are certainly cases running the course (about three years) having the symptomatology, bulbar paralysis, etc., of what Charcot calls lateral amyotrophic sclerosis, in which the symmetrical lateral sclerosis is not the primary lesion (my own and Leyden's cases).

Secondary changes are seen in the lateral white matter in cases of infantile spinal paralysis in which the lesion is an acute myelitis of the anterior horns. No special attention appears to have been paid to this condition, nor has the question arisen why it is there. It must be said that Charcot states that he has never seen a case in which the myelitis of the anterior horns caused secondary degeneration of the lateral columns, but he does not appear to think that it is impossible.

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* *Etude sur le Sclerose laterale amyotrophique.* 1877.

One point appears to me of great importance in the microscopic examination of these cases, and that is a careful observation and record of the degree of change in the different regions (anterior horns and lateral columns). The conclusion from all this is evident, that the symptomatology and the interpretation of the pathological changes seen in the so-called lateral amyotrophic sclerosis of Charcot will have to undergo modifications, and cannot be considered so absolute as Charcot has laid it down.

EXPLANATION OF WOOD-CUTS.

The shaded regions indicate the sclerosis.

Fig. 1.—From cervical enlargement, showing distribution of sclerosis in lateral white matter.

Fig. 2.—From dorsal region; it will be observed that the sclerosis does not occupy exactly the same region as in Fig. 1, but keeps very near to the posterior horn. In some sections from this region the sclerosis extended pretty well forward, and as far as the anterior border of the anterior horn, and keeping close to the grey matter.

Fig. 3.—From lumbar region. Here the sclerosis is very limited in extent and is well indicated by wood-cut.

Fig. 4.—From section through middle of olivary bodies; here the sclerosis is of an exceeding slight character. Fig. *a*.—Sclerosed anterior pyramid. Fig. *c*.—Nucleus of hypoglossus. Fig. *b*.—Sclerosed region in district which Meynert considers as belonging to the lateral regions of the spinal cord; also a light band of sclerosis extending from this down almost to the sclerosed anterior pyramid.

Fig. 1

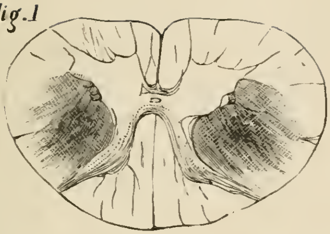


Fig. 2.

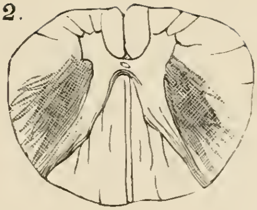


Fig. 3.

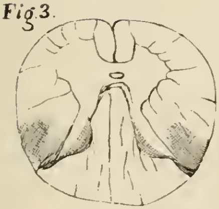
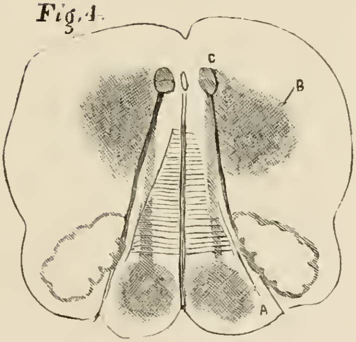


Fig. 4.



ART. V.—CEREBRAL THERMOMETRY.*

BY LANDON CARTER GRAY, M. D., BROOKLYN, N. Y.

IT is well known to all neurologists that M. Paul Broca, whose reputation as an investigator has justly spread over two hemispheres, read a paper on Cerebral Thermometry before the *Association française pour l'avancement des Sciences*, at its meeting on the 30th of August, 1877.† Upon perusal of the report of this communication, I was greatly impressed with the possible value of the subject as a diagnostic aid in cerebral affections. I soon afterward determined to repeat the experiments of the illustrious Frenchman upon a more extended scale, and this paper contains the results of such repetition.

I regret that I have been obliged to content myself with a mere abstract of M. Broca's paper, not having been able to obtain a full copy, although I have sent to Paris for the purpose. This fact will excuse any inadvertent injustice that I may happen to do the gentleman in not crediting him with more fullness of detail than has been attributed to him in the report which I possess. I have thus not been exactly positive as to what variety of thermometer M. Broca used, although, from the meagre description of the reporter, I have presumed that he employed one similar to the one which I have adopted, and which is well known as the Seguin surface thermometer (Fig. 3). As all American physicians are aware, this consists of a button-like bulb, about three-eighths of an inch in diameter, from which emerges an upright registering tube about four inches in length. The first set of these, although carefully made for me by Messrs. Tiemann & Co., proved to be eminently unsatisfactory, and for two reasons: they were not self-registering, and moderate pressure upon the bulb was found to elevate the registering column slightly over one degree. The necessary manipulation of the instruments, of which I shall speak presently, rendered these defects fatal to accuracy. This easy response to moderate pressure upon the bulb is, I am led to believe, a very general although unrecognized fault with thermometers of this variety. As a curiosity, I have brought one with me to-day, made by some foreign manufacturer, in which, as you see, pressure upon the bulb alters the scale 15 degrees. I rejected, therefore, the observations already made, and ordered a new set. After repeated and careful experimentation, Mr. A. Bayer, by whom these thermometers are made for Messrs. Tiemann & Co., succeeded in completing a set, in some of which moderate pressure had no appreciable effect, while in others such pressure would cause the scale to rise about one-fifth of one degree; and these were made self-registering. Before placing the thermometers in position, I have made a practice of shaking the register down 10 to 12 degrees below the probable temperature of the part to be registered, thereby avoiding all possible error from pressure. I have taken great care that these instruments should be also perfectly accurate as registers of heat. They were compared with a standard thermometer in the possession of Mr. Bayer, which had been seasoned for three years in the rough, then carefully and repeatedly graduated by a standard thermometer of Mr. James Green, the well-known maker of scientific apparatus, which latter is identical with Casella's standard; and from time to time since their manufacture my instruments have been again tested.

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* Although this article has already appeared in a medical journal in this country, we reproduce it here, as of interest to neurologists. It will be followed by another from the same author in the April issue of the JOURNAL. —EDITORS.

† *Progrès Médical*, 1877.

For holding the thermometer upon the head I employ a ribbon, such as is used for a laryngoscopic mirror, or a morocco band (Fig. 1), either of which is perforated throughout its length by holes of proper size to snugly embrace the tube. This ribbon or band encircling the head, upon a level with the eye-brows and the temporal plane just above the ears, and being buckled across the forehead, I station thermometers in it at the points indicated by M. Broca, as follows: One on each side somewhat back and above the commencement of the external angular process of the frontal bone, one on each side just above the ear, and on each side of the occiput. The two anterior points I have termed the Frontal Stations, the two above the ear the Parietal Stations, and those posteriorly the Occipital Stations. In this manner the frontal, the parietal, and the occipital lobes are fairly approached. But these temperatures indicate only the temperature of the sides of the brain. It has seemed to me important, as well as feasible, that the temperature of the motor region of the vertex should also be ascertained. With this end in view, I have profited by the recent researches in cranial topography of MM. Broca,* Heftler,† and Féré.‡ According to these writers the fissure of Rolando, which may be regarded as at the centre of the motor region of the vertex, abuts upon the great longitudinal fissure at a distance of 47 to 48 millimetres, or about $1\frac{7}{8}$ inch, to the rear of the coronal suture. As the coronal suture, however, is not easy to distinguish through the scalp in the living person, I have selected the furrow formed by the junction of the nasal bones with the nasal process of the frontal as a more salient point of departure, and have determined its distance from the coronal suture by measurement of 24 skulls. The average was found to be 5 inches, the maximum $5\frac{3}{8}$ inches, the minimum $4\frac{5}{8}$ inches. This average, or a deviation from it of not more than $\frac{3}{8}$ of an inch, was maintained in 95.82 per cent. of the measurements. Adding together the calculations of the French authorities as to the distance of the fissure of Rolando from the coronal suture, and my own as to the distance of the coronal suture from the junction of the nasal bones with the frontal, the average distance of the fissure of Rolando from the fronto-nasal junction is ascertained to be about $6\frac{7}{8}$ inches, the deviation being so slight as to be practically of little moment. Mapping out the fissure of Rolando in this manner, I have attached by loops two ribbons or bands to the ribbon or band encircling the head, in such a manner that one may be buckled across the vertex anteriorly and the other posteriorly to the fissure (Fig. 1). In each of these bands I station two thermometers, one on each side of the median line of the head, and about an inch apart, so that a square is thus formed, roughly tracing the outline of the motor zone of the vertex, and at whose four corners are situated the Vertical Stations. These bands or ribbons are of the same material as the lateral headband, and similarly perforated.

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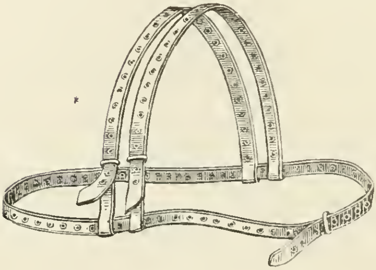
* "Sur la topographie crânio-cérébrale," *Rev. d'Anthrop.*, t. v., No. 2, 1876.

† "Circonvolutions cérébrales chez l'homme, et leurs rapports avec le crâne," par Ferd. Heftler, Dissert. Inaug. à l'Acad. Med.-Chir. de Saint-Petersbourg, 5 mai, 1873.

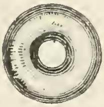
‡ "Note sur quelques points de la topographie cérébrale," par Ch. Féré. *Bull. Soc. Anat.*, 24 déc., 1875.

FIG. 1.



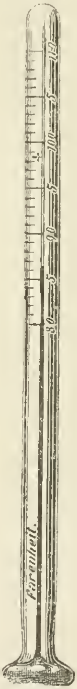
HEAD-STRAP.

FIG. 2.



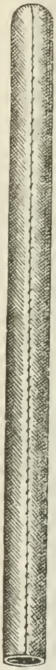
PAD.

FIG. 3.



THERMOMETER.

FIG. 4



FINGER.

The instruments have been carefully protected against atmospheric variations by a small silk pad lined with wool (Fig. 2), and which has an orifice so as to permit of its being slid over the tube down to the upper surface of the bulb, as well as by a silk finger (Fig. 4), also lined with wool, which is pushed over the tube.

It has been my habit to arrange the head-gear in this manner : I first buckle the band around the head. Sliding a pad over each thermometer, passing the tube through a perforation opposite the proper place, and then covering it with a silk finger, I adjust an instrument at each Frontal, Parietal, and Occipital Station. I now fasten the vertical bands in place, and, observing the same precaution as to pads and fingers, I arrange the four thermometers as I have described. The instruments remain on the head for twenty minutes ; and then, gently and carefully removing the silk fingers, without at all moving the bulb from the scalp, I read off the markings.

M. Broca's observations were made on twelve internes and dressers, who were nearly of the same age, the same intellectual development, and placed very nearly in the same circumstances. For purely scientific or physiological purposes, this similarity of age, intellect, and surroundings, was undoubtedly of great advantage. From a utilitarian, diagnostic point of view, however, this feature is of equally great disadvantage, because it would be impracticable to select our patients so nicely and critically. At the outset, therefore, I resolved to take persons as they came, simply being careful to avoid any extremes, as of great emotion, mental excitement, or prolonged slumber, and that there should exist no appreciable ill-health. My observations were made upon 92 of the medical students in attendance at the Long Island College Hospital, as well as upon 10 physicians, numbering in all 102 males. The age has been on the average 28.94, the maximum 50, the minimum 18. Of this number I am unable to make use of 40 for absolutely accurate calculations, because of a defect in my thermometers of somewhat less than one-quarter of one degree, which was unnoticed for several days, and which I cannot eliminate in any other way than by this wholesale rejection. For calculations, however, that are merely comparative, and wherein this defect would be harmless, I shall employ these imperfect observations without hesitation. In all of my cases I have also taken the temperature of the mouth with an accurate thermometer. The Fahrenheit scale has been used in preference to the centigrade, as it is more familiar to Americans.

The average of the Left Frontal Station was 94.36°, that of the Right being 93.71°, the difference being .65°. At the Left Parietal the average was 94.44°, while at the Right it was 93.59°, the difference being .95°. The average of the Left Occipital Station was 92.66°, that of the Right 91.94°, showing a difference of .72°.

Thus, it will be perceived, the left lobes possess a higher temperature than do those of the right, the difference amounting on the average to .74°. Moreover, the range from maximum to minimum at the lateral stations on the right is larger than it is on the left, being on the average 6.25° on the left and 7.58° for the right, showing a preponderance in favor of the right of 1.33°. It will also be remarked that the temperature of the brain decreases posteriorly, with the single exception of the Left Parietal Station, of which the average is .04° higher than that of the next highest point, the Left Frontal. In this last particular my observations differ from those of M. Broca. Has the connection between the third left frontal convolution and the memory and faculty of language anything to do with this phenomenon? I am inclined to think that it has. M. Heftler* states that the lower portion of the fissure of Rolando is 28 millimetres, or about $1\frac{1}{11}$ inch, to the rear of the fronto-parietal suture. It has been at or rather anteriorly to this point that my Parietal Stations have been situated, so that they have actually been over or in close proximity to the third frontal convolution at its upper and posterior part, where it is bounded by the lower portion of the fissure of Rolando. The memory of words and the faculty of articulate language, to which the structural integrity of the third left frontal convolution is known to be necessary, are so interwoven with all forms of mental activity, as thought and the emotions, that it is by no means a bald hypothesis to suppose that this convolution may have a more incessant and complicated function to perform than any other portion of the brain.

The maxima and minima of temperature at the lateral stations were as follows:

**Op. cit.*

	Maximum.	Minimum.	Difference.
Left Frontal	97°	91°	6°
Right Frontal	97°	89.75°	7.25°
Left Parietal	96.50°	90°	6.50°
Right Parietal	97.50°	89.50°	8°
Left Occipital	95°	88.75°	6.25°
Right Occipital	95.50°	88°	7.50

The average range from the maximum to the minimum was 6.91°.

The average of the whole of the left side of the head was 93.83°, of the right 92.92°, there being a difference of .91°. The maxima and minima ran thus :

	Maximum.	Minimum.	Difference.
Left side	96°	90.83°	6.83°
Right side	95.25°	90°	5.25°

The average range of the sides of the head from maximum to minimum was 6.04°.

The average of the whole head, exclusive of the vertex, was 93.51°, the maximum 96.09°, the minimum 89.75°, the range 6.34°.

The average of the temperatures taken in the mouth was 99.68°, the maximum 100.50°, while the minimum was 99°. This average is somewhat higher than it is generally supposed to be, and yet it is in striking accord with that obtained by Dr. J. Davy,* in his series of researches upon 114 subjects of both sexes, of different ages, among various races and latitudes, and with various atmospheric temperatures. His mean was 100°, the highest temperature 102°, the lowest 96.50°. It is a curious coincidence that the mean temperature of the atmosphere in his investigations was about the same as in mine. Of this I feel quite positive, although I have no exact notes of this factor in my own cases.

The temperature of the vertex was taken in persons whose

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*Carpenter's *Physiology*, edited by Francis G. Smith, M. D. : Philadelphia, 1876, p. 529.

hair was decidedly thin, or who were bald. The hair at the sides need only be encountered at the Occipital Stations. At the brow and above the ear the thermometer can always be placed directly upon the skin; and even at the occiput the hair can be pushed to one side to so large an extent as to interfere but slightly with the result, as I shall presently endeavor to show. At the vertex, however, the hair is usually very much thicker, longer, and more difficult to dispose of, rendering observations of the kind of which I am treating extremely variable and uncertain. I regret to state that for these reasons my cases have been limited to 13—too small a number upon which to base positive conclusions, but which I hope to supplement at some future day with others. I have not thought it worth while to detail the temperature at the four individual stations, nor of those to the left and right of the great longitudinal fissure, as they are all too closely approximated to render this procedure of any value, but rather to calculate the average of the whole vertical quartette. This was 91.67°. The maximum stood at 94.50°, the minimum at 83.37°, the range from maximum to minimum being 6.13°.

The temperature of the whole head, inclusive of the vertex, was 92.66°, the maximum 95.02°, the minimum 90.67°, the range from maximum to minimum 4.35°.

I have endeavored to ascertain what difference was effected at the Occipital Stations by the hair. I have, therefore, compared the temperature at these spots of thirteen thin-haired persons with the temperature at the same points in thirteen thick-haired individuals. In those with thin hair the average on the left was 92.62°, standing at 92.11° on the right. The thick-haired possessed an average on the left of 92.19°, on the right of 91.38°. The average elevation of temperature obtained in the thin-haired above those whose hirsute covering was more abundant was .58°, or slightly above one-half of one degree.

The effect of mental exercise upon the temperatures has been exceedingly variable. My observations only number four, and I do not cite them to demonstrate a general law, which can only be established by more extended study, but simply to contribute in some slight degree to this study. In the first of my subjects, a very intelligent physician, after reading ten minutes, the only rise was one of .50° at the Right Parietal Station. In the second person, a young student, ten minutes' reading produced an elevation of .75° at the Left Parietal Station, and one of .50° at the Right Frontal. My third case, an eminent physician of an active and profound mind, was tested after a vivacious lecture, and yielded the following figures: A decrease of 1.67° at the Left Frontal, an increase of 2.50° at the Left Parietal. On the right side there was an increase at each of the three Stations amounting to .67° at the Frontal, to 1° at the Parietal, to 2.50° at the Occipital. Yet the average of the whole left side amounted to .98°, whereas that of the whole right descended .28°. The average of the whole head, however, exclusive, of course, of the vertex, which was not taken into account for this purpose, was raised .25° by the lecture. The fourth observation varies still more remarkably. It was made upon a distinguished professional gentleman, who is a well-known writer and an active lecturer, and was taken after a lecture delivered in the full tide of a medical term. On the left side the Frontal and Occipital Stations fell .25° and 2° respectively, and the Parietal rose .50°. The Right Frontal rose .50°, the Parietal 1°, while the Occipital fell off .75°. The average of the left side was .69° higher after the lecture, whereas that of the right was .25° lower. But the average of the whole head was elevated 1.17°. The averages computed from these figures showed, nevertheless, that the psychical labor had increased the actual evolution of heat in general, however much it might vary at particular points, the average of the whole head having ascended .14°, the average of the left side .19°, the average of the right side .07°. In this connection I desire to call attention to a fact lending support to the theory which I have advanced in regard to the relative functional activity of the third left frontal convolution. In three of these four cases there was an elevation of temperature at the Left Parietal Station of from 2.50° to .50°; the average ascent of the four subjects was thus .89°. In other words, there was very generally, so far as these scanty figures show, an increased amount of caloric given off in the neighborhood of the third left frontal convolution, and the average of this amount was greater than the average of the increase at any other point.

These observations tally in many respects with those of M. Broca. He states the average of the left and right sides to be respectively 93.20° and 93.02°, while my figures are for the same points 93.83° and 92.92°, standing .63° more on the left and .10° less on the right. Again, according to him, the average of the whole head is 92.87°, and the maximum and minimum are affirmed to be each 94.75° and 91.04°. I found this average to be 92.66°, or .21° lower than he did, and the maximum and minimum to be 95.02° and 90.67°, or .27° higher in the former and .37° lower in the latter. These variations are so trivial that they may readily be explained by the great disproportion between the number of M. Broca's cases and my own. Indeed, to my mind, the difference is so slight that I even deem it worthy of being regarded as evidence of the remarkable uniformity of the cerebral temperature. I cannot look upon it as a mere coincidence that the temperature of twelve young men, who were selected for experiment with great precautions as to similarity in age, intellect, and surroundings, should approximate so closely to the temperature of 102 individuals, who were purposely selected, so to speak, with carelessness.

It will have been observed that the range from the maximum to the minimum temperature is a large one when compared with that of the axillary or buccal temperature. For instance, in my cases the buccal temperature ran over a scale of 1.25°, the maximum rising to .58° above, while the minimum was .68° below the average. In the cerebral temperatures, on the other hand, the average range at the lateral stations was 6.91°, while at the vertex it was 6.13°. *A priori* it might well be supposed that the production of heat within the cerebrum would fluctuate within much wider limits than it would in the body. The wonderful complexity of the function of the brain; its almost ceaseless activity: the ease with which it is animated or depressed by innumerable internal and external influences: the manifest provision for normal fluctuations of the vascular supply within comparatively wide limits, as is evidenced in the greater amount of contractile fibre in the cerebral arteries relatively to the peripheral vessels, as well as in the arterial expansion which is permitted by the peri-vascular spaces: all point to this organ as one in which the molecular changes proceed with unrivaled vivacity and variability, and in which the evolution of heat whould be characterized by equal rapidity and variation. Can we then in the face of this large range of the temperature of the normal brain from maximum to minimum, make any diagnostic use of the thermometer? Let the ensuing figures answer this question. In 61.84 per cent. of the observations, the temperature stood either at the average, or from 1.13° above to 1.14° below. In 77.95 per cent. of the cases, the temperature was either at the average, or from 1.64° above to 1.70° below. In 86.23 per cent. of the personages examined, the temperature was at the average, or from 2.13° above to 2.26° below. In 64.33 per cent. of the cases, the temperature at each of the lateral stations on the left was higher than that of the corresponding stations on the right. In 10.33 per cent. the temperature at each of the lateral stations on the left was the same as at the corresponding stations on the right. In 25.33 per cent. of the cases, the temperature at the lateral stations on the right was higher than at the corresponding stations on the left. The average of the whole of the left side was higher than that of the right in 70 per cent. Finally, the average of the whole of the right side was higher than that of the left in 30 per cent. *In other words, when the temperature goes beyond one and a half degree above or below the average, there will be only* 22.05 *persons in every hundred in whom it would be normal; when the temperature surpasses about two degrees above or below any average, there will be only* 13.77 *individuals in a hundred in whom this temperature would be normal; and only* 25.33 *persons out of a hundred will display a higher temperature at any station on the right side than at any corresponding station on the left.*

In view of all these facts, I feel myself warranted in drawing the following conclusions:

1. The average temperature of the Left Frontal Station is 94.36°, the Right being 93.71°.

2. The average temperature of the Left Parietal Station is 94.44°, the Right being 93.59°.

3. The average temperature of the Left Occipital Station is 92.66°, the Right being 91.94°.

4. The average temperature of the left side of the head is 93.83°, the right being 92.92°.

5. The average temperature of the whole head, exclusive of the vertex, is 93.51°.

6. The average temperature of the motor region of the vertex is 91.67°.

7. The average temperature of the whole head, inclusive ot the vertex, is 92.66°.

8. If there be an alteration of temperature at any of the lateral stations of more than one and a half degree above or below the average temperature of such station, this fact will justify a *suspicion* of abnormal change at that point.

9. If there be an alteration of temperature at any of the lateral stations of more than two degrees above or below the average of such station, this fact will constitute *strong evidence* of the existence at this station of abnormal change.

10. In proportion as the alteration of temperature at any individual station is increased or decreased beyond the figures just mentioned, in exact proportion will the strength of the evidence be increased as to the existence of abnormal change at that station, until, the maximum or minimum having been passed, the evidence will become almost conclusive.

11. Should it so happen that such elevation of temperature above the average should be at any lateral station on the right, causing a rise at this point beyond the average temperature at the corresponding station on the left, this would strengthen the suspicion or the evidence.

12. These remarks apply with equal force to the average for the whole of either side, as well as to the average for the whole head.

13. It is necessary to the validity of these conclusions that the contemporaneous bodily temperature should be normal, or that there should be a marked disproportion between it and the cerebral temperatures.

Through the kindness of my friend, Dr. Frank W. Rockwell, of Brooklyn, I have been enabled to test the value of these deductions in a very interesting case of tumor of the brain,* which I was courteously permitted to report to the New York Neurological Society, at its meeting on June 3d of this year, and of which Dr. Rockwell will soon publish a full account. The patient was a female, aged thirty-four. There was present a typical "choked disk," marked pain in the temple and brow, becoming unbearable in paroxysms, nausea, vomiting, ptosis, paralysis of the ocular muscle. The first paroxysm of pain came on January 21st. The bodily temperature ranged near the normal. Upon these symptoms a diagnosis of intra-cranial tumor was made, probably situated at the base. Placing my thermometers upon the head, I ascertained the temperature at the different stations to be as follows:

	Left.	Right.
Frontal	96.75°	98.33°.
Parietal	95°	99.75°.
Occipital	96.75°	100.50°.

The average of the two sides, if calculated, will be found to be 96.16° on the left, on the right 99.52°, the average for the whole head being 97.84°.

The rise above the normal averages is startlingly apparent. At the Left Frontal Station it was 2.39°; at the Left Parietal, .56°; at the Left Occipital, 4.09°; at the Right Frontal, 5.12°; at the Right Parietal, 6.16°; at the Right Occipital, 8.56°; while the average of the left side had mounted above the normal 2.33°, the right side 6.60°, and the average of the whole head 4.33°!

This particular observation was taken as I was at the outset of my study of the subject, and was made with my first set of thermometers, which, as I have already stated, were defective. I have satisfied myself, however, that the defect amounted to but a little over one degree. If, therefore, from these figures one and a half degree be deducted, all fear of error may be dismissed; and yet the increase is unmistakable. About this date (March 4th), I wrote Dr. Rockwell: "I shall certainly expect to see inflammatory changes from the base of the fissure of Sylvius backward along the occipital lobe, as well as that these changes shall be spread around the base of the fissure." The patient died March 16th. At the *post-mortem*, at which, much to my regret, circumstances prevented my attendance, there were present Drs. Rockwell, Arthur Matthewson, and E. S. Bunker. The meninges were found apparently normal, with the exception of a slight congestion. At the base of the brain the membranes and skull were to all appearances healthy. But a soft, jelly-like tumor, the size of a hazelnut, was found between the horizontal or posterior branch of the fissure of Sylvius and the first temporal fissure, while the whole of the right occipital lobe was converted into a colloid, extremely vascular mass, which gave way under examination, this degeneration also extending anteriorly to the tumor as far as the fissure of Sylvius. There was no apparent disease except at these points. Upon microscopical examination, I ascertained the tumor to be a typical glioma, thickly strewn with small extravasations of blood.

This is, I believe, the first case on record in which a diagnosis of the locality of an intra-cranial morbid growth has been made with a thermometer, and afterward verified by a *post-mortem* examination.

I cannot resist the conviction that these data are sufficient to enable the profession to employ the thermometer with profit in the diagnosis and localization of cerebral affections. I trust that experience will substitute proof for my conviction. I am now engaged in the study of the pathological aspect of this question. My material is not yet ripe for publication, but I expect at some future day to be able to communicate to the profession further details as to the exact possibilities of this subject.

Neurological Correspondence.

ABSTRACTS FROM THE PROCEEDINGS OF THE NEW YORK NEUROLOGICAL SOCIETY.

By W. J. Morton, M. D., Secretary.

Messrs. Editors: — During the last six months the proceedings of the society have been full and interesting. What follows represents but a part of its work, since several papers have been, or are to be, published in this journal, and need therefore be only alluded to here.

At the April meeting Dr. T. A. McBride read a paper entitled "Digiti Mortui;"* specimens of a case of progressive muscular atrophy were shown by Dr. J. C. Shaw;† and the officers for the ensuing year were elected.

At the May meeting Dr. Seguin, the retiring president, made a few remarks, and introduced Dr. Shaw, the president elect. Dr. Seguin read a paper on a case of locomotor ataxia, followed by general discussion.

At the June meeting Dr. Landon Carter Gray gave the history of a cerebral glioma. The symptoms were: a typical "choked disk," marked pain in the temple and brow, becoming unbearable in paroxysms, nausea, vomiting, ptosis, paralysis of the ocular muscles. The first painful paroxysm began on Jan. 21st. Death occurred on the 16th of the ensuing March. Patient was a female, aged 34. The tumor was soft, jelly-like, of the size of a hazelnut, situated between the horizontal or posterior branch of the fissure of Sylvius and the first temporal fissure on the right side, while the whole of the occipital lobe was converted into a colloid, extremely vascular mass, which gave way under examination. This degeneration also extended anteriorly to the tumor as far as the mouth of the fissure of Sylvius. The meninges were normal.*

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* This will appear in the April number of the Journal of Nervous and Mental Disease.

† Published in the present number.

Dr. Gray also reported a case of large tumor of the medulla oblongata, of which he possessed an imperfect history, having never seen the patient before the autopsy. Nine or ten weeks before death, symptoms began with intense occipital pain, although there was a specific taint of four years' standing, which had been manifested by occasional slight disturbances of health. After a week's duration, the occipital cephalalgia remitted, and the patient resumed work, though not with his usual vigor. It soon returned, and was speedily followed by partial paralysis of the feet and legs, this paralysis ascending until the four extremities were involved, at the same time increasing locally in severity. At the time when the patient came under observation, three days before death, there was complete paralysis of the four extremities, and the muscles of respiration were laboring heavily. Head and face free from paralysis, and voice normal, though he preferred whispering to save his breath. He was somewhat emaciated, and skin was dry and harsh. Bladder and rectum paralyzed, latter having been in this state for three weeks. Urine was purulent. Rectum was found dry as the skin, great rolls of epithelium coming away, but nothing resembling fecal matter. Was perfectly rational, though somewhat dull. Death from failure of respiratory muscles. The heart continued to beat quite as strongly as it had done for days up to and even some half-dozen beats beyond the cessation of respiration. Autopsy: An irregularly conical tumor was found on the posterior surface of the oblong medulla, extending from the cerebellum to 5½ centimetres below, and pressing upon the lower portion of fourth ventricle. The growth had been entirely from the dura mater, no attachment to the medulla having taken place.

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* This case was mentioned by Dr. Gray in his article on "Cerebral Thermometry," read before the American Neurological Association, and published in the *New York Medical Journal*, Aug., 1878. The site of the tumor was diagnosed by him with his cerebral thermometers, and it is claimed to be the first thermometrical diagnosis on record of the locality of a cerebral tumor.

The tumor was irregularly lobulated, being 2½ centimetres thick and 5½ wide at the base of the cone, which was above, and about ½ centimetre thick and 1½ wide at the apex. It had flattened out the posterior surface of the medulla to some extent, and there were shallow depressions on this surface and on the floor of the ventricle formed by the lobules. The growth was hard, resisting, fibrous to the touch. Microscopical examination showed it to be a fibro-sarcoma.

Dr. Seguin presented some specimens of sections from the spinal cord of an anencephalous fœtus.

Dr. Hammond then read a paper "On Obscure Abscesses of the Liver; their association with Hypochondria, and their Treatment." Beginning with the proposition, that abscess of the liver may exist without exciting suspicion, either by any marked general or local symptoms, the reader proceeded to sustain this point by numerous citations of authors and of instances, and then passed at once to the recital of the particulars of several cases which had come under his own observation. Omitting many details of these cases, five in number, they may be thus briefly summarized:

Case I.—B. B., aged 45, suffering from spinal paralysis, had injured his right side by a fall. There was never any decided pain in the part injured nor enlargement, and no fever or signs of exhaustion appeared. Gastric derangement, some pain at times in the right shoulder, and very marked hypochondria, however, existed. It was thought that fluctuation could be detected over the anterior surface of the liver. The needle of an aspirator was introduced through the tenth intercostal space, rather behind a line let fall from the middle of the right axilla, and fifteen and one-half ounces of pus were evacuated. No unpleasant symptoms followed the operation; the patient's general health as well as his mental condition greatly improved, and he remains well except as regards the paralysis of the lower extremities, from which he had previously suffered.

Case II.—E. P. R., aged 70, had been for several years under treatment for cerebral symptoms, and later had a slight seizure, apparently of cerebral hemorrhage, not followed, however, by any permanent paralysis. In August, 1876, after a considerable interval, during which he had regained a good degree of health, he again came for consultation, complaining of great depression of spirits, irregular action of the heart, gastric derangement, alternate constipation and diarrhœa. The conjunctivæ were of a slight yellow tinge, though no bile was discovered in the urine. There was no enlargement of the right side of the abdomen, no distinct pain in the liver, and but slight tenderness on pressure. Nor could fluctuation be detected with any certainty.

The fear of death and other morbid apprehensions were, however, so great that it was determined to aspirate the liver. A small needle was introduced, as in the previous instance, and eight and a half ounces of pus were evacuated. The patient made an excellent recovery; the mental symptoms disappeared at once, and his general health became better than it had been for years. There has never been since the operation any approach to hypochondria.

Case III.—J. N. C., aged 48, suffering from cerebral hyperæmia, which was at first relieved by removing a small growth from the external meatus of the ear. In a month however, these symptoms returned. He was also depressed in spirits, the stomach was deranged, nausea was frequent, there were irregular action of the heart and a peculiar livid appearance of the face and neck. Insomnia remained a marked symptom. Under treatment amendment occurred, but after a few months the patient again returned, worse than he had yet been. Insomnia was distressing; he had unpleasant dreams when he did sleep; he was apprehensive of impending death, insanity or paralysis. There were pain in the head, a sensation of weight at the vertex, vertigo, irregular and at times intermittent action of the heart, constrained respiration, and also the peculiar lividity previously noticed. Dyspepsia, constipation, and a slight yellow tinge in the conjunctivæ were also present. Slight but distinct evidence of bile in the urine was discovered.

Examination of the liver failed to show any enlargement or tenderness. It was thought that fluctuation could be detected. Inquiry revealed the fact that the patient had several years previously suffered from malarious attacks, and was now living in a malarious region.

On the 21st of August the needle of a hypodermic syringe was introduced through the ninth intercostal space, and pus obtained. On the 23d an aspirating needle was introduced, and ten ounces of highly offensive brownish-colored pus evacuated. The same needle was then reinserted at the spot where the hypodermic syringe had been previously introduced, and four ounces of pus, quite free from odor, obtained. The patient regained excellent health, his cerebral symptoms entirely disappeared, his complexion became clear, his stomach and bowels recovered their normal functions, and he is now able to transact an immense business.

Case IV.—W. J. S., aged 30. Three years previously was treated for symptoms indicative of cerebral hyperæmia, with gastric disturbance. Persistent wakefulness was a marked symptom. His general health was bad, his body emaciated and his urine loaded with urates. He improved under treatment but was subject to relapses, apparently due to over mental exertion. In April of the present year he was again seen. His symptoms were pain in the head, insomnia, and hypochondria of a very decided form; also dyspepsia characterized by pain, burning, nausea and vomiting. The urine contained a large excess of phosphates, but no bile. The conjunctivæ were tinged and the complexion sallow. Emaciation was extreme. Examination of the liver, owing to the thinness of the patient's abdominal walls, was effected with ease, but no enlargement or prominence or tenderness of that organ could be detected, nor was pain complained of in its region. There was however distinct fluctuation. The patient agreed to an operation, and accordingly the smallest-needled fan aspirator was introduced at the eighth intercostal space, taking care to making the puncture valvular. Eight and a half ounces of odorless pus were evacuated. The patient immediately began to improve in appetite and spirits, his dyspepsia disappeared, he slept well, the pain in his head ceased, his complexion became clear, and in a few days he returned home quite recovered.

Case V.—F. C. S., aged 48. His symptoms were pain in the head, occasional vertigo, wakefulness, great mental depression, dyspepsia, and other indications of cerebral hyperæmia. Under treatment he improved greatly, except in the one point of mental depression. After an interval of six months of fair health, all his old symptoms again made their appearance, the hypochondria especially being present to an alarming extent. He had lost all interest in life, though at the same time full of the most gloomy apprehensions in regard to himself. He was certain that his disorder must end in death or insanity, and he did not care which. Of the diagnosis of cerebral hyperæmia there was no doubt, but added to this was a degree of hypochondria approaching to insanity. The gastric disturbance was great, there was no appetite, he suffered severely from alternate diarrhœa and constipation, and the complexion was sallow. After careful and thorough examination, no signs of disease of the liver could be detected by abdominal examination, neither by the operator himself nor by the two medical gentlemen present.

It was agreed that there were absolutely no local signs of abscess, nor any such general symptoms as are commonly supposed to indicate the disease in question. With previous experience in mind, however, it was determined to operate with the aspirator, which was accordingly introduced through the eleventh intercostal space, at a point two inches in front of a line let fall from the middle of the axilla. At a depth of two inches pus flowed, and nine ounces of odorless, creamy-looking pus were obtained. For the first time in many weeks the patient slept at night. As regards all his symptoms, the change for the better became well marked, and there is little doubt that complete recovery will result.

The points which the reader considered established by the foregoing cases are :

1. That hepatic abscesses are probably much more common with us than is generally supposed.

2. That they may exist without any local symptoms or such general disturbance of the system as is commonly regarded as indicating their presence.

3. That they may be associated with hypochondria and other evidences of cerebral disturbance.

4. That they should be opened at the earliest possible moment, and without waiting for adhesions to form between the liver and the abdominal wall.

5. That the proper place for performing the operation of aspiration is in one of the intercostal spaces.

6. That the operation by aspiration is free from danger.

7. That in all cases of hypochondria or melancholia, the region of the liver should be carefully explored, and that even if no fluctuation be detected or any other sign of abscess be discovered, aspiration, being a harmless operation, should be performed.

8. That if pus be evacuated, the operation may be expected to be followed by a cure of the mental disorder as well as by the preservation of the life of the patient from the probably fatal consequences of hepatic abscess.

9. That if no abscess be found the patient will, at least, be no worse off than he was before.

Relative to the third of these propositions, the association of liver disease or disorder, including abscess, with hypochondria may be readily granted; but there are beyond this points of importance. What, for instance, is the cause of the abscess of the liver, in these cases? The reader was inclined to think that they are due to the brain disturbance in the first instance, and that by their influence the cerebral symptoms were altered and intensified.

It is well known, also, that congestion and inflammation of the lungs may be the direct result of cerebral hemorrhage, and that the mal-nutrition of the skin leading to the production of ulceration, is sometimes a consequence of a like cause. There are, also, many other examples that might be adduced, going to show the influence of lesions or disease of nerve centres over the nutrition of distant organs. There are, therefore, strong physiological and pathological reasons for ascribing these hepatic abscesses to the direct influence of brain diseases, especially hyperæmia, by which the nutrition of the liver is impaired, and destruction of a certain portion of its tissue effected. That they are invariably attendant upon such a condition is not to be asserted. Doubtless there are pathognomonic signs of their presence which only further experience can discover. At present we move in the dark, or at least in a very obscure atmosphere. But in the harmlessness of the operation necessary for their cure we have a factor which cannot but aid in our further enlightenment.

At the October meeting Dr. Seguin presented a case of Progressive Facial Atrophy, as follows:

Mr. President and gentlemen:—The patient who is before you is the bearer of so rare a disease, one so little understood, that every instance of it is well worthy of study. The following is a summary of a history of the case:

Delia H., ten years of age, has enjoyed general good health, and comes of sound parentage. When she was five years of age her mother noticed a greenish spot "like a ringworm" on the left cheek, midway between the malar bone and the angle of the mouth. There were no pains in the face at this, or at any subsequent time. Very gradually (perhaps in the course of one year), the cheek began to sink in, and the mouth to become crooked, being drawn upward and to the left. No other spots have been noticed. The disease has slowly progressed without pain until the present time. The physical and mental development of the child have progressed unimpaired.

Present condition: The child is well-grown, and healthy-looking. The face is much disfigured on the left side by an atrophy of a large part of the cheek. The mouth is drawn upward and to the left; its upper red border is small and almost linear. From a distance the skin appears healthy. The region lying between the malar eminence and the mouth is the seat of a deep excavation, in which the volume of half a walnut would have to be placed in order to imitate the rotundity of the other cheek. Closer examination shows the following points: A whitish atrophic spot exists in the centre of wasted region, about the size of a five cent silver coin; but the rest of the skin of the head and face is healthy. All the tissues of the cheek are much wasted, and its thickness is nearly one-third less than that of the opposite side. The left upper segment of the lip is excessively thin, but these soft parts are in no wise abnormally adherent to the bone. The remainder of the face on the left side is rather thinner than on the right, but no positive atrophy exists except in the region above described. The tongue and soft palate are normal. The left superior maxilla is, however, the seat of marked wasting; its alveolar border not being more than half as thick as that on the right side. The upper teeth are more regular on the diseased than on the healthy side; they are of good size and well preserved. The space between the two upper middle incisors is abnormally great. The whole set of teeth is irregular, but not Hutchinsonian or specially rachitic. The anterior part of the hard palate is deeper (atrophied) on the left side. The lower jaw is normal. The pupils are equal and normal. Voluntary movements are almost perfectly full and free on the atrophied side of the face.

Sensibility: The points of the esthesiometer are distinguished at the following distances: on the forehead, at 15mm. on each side; on either cheek at 12 and 12mm.; on the lower cheek and over the lower jaw, at from 6 to 10mm.; on the red surface of the lips at 2mm.; there being no difference between the two sides even on the thinnest part of the left upper lip. Pricking is well felt everywhere on both sides of the face.

Faradic reaction in the facial nerve: The right and left nerves are substantially equal, giving reaction with the minor current and four inches of cylinder of Kidder's battery. Applying the current to the muscles on the outer and on the inner surfaces of the cheek, gives the same result, *i. e.*, irritability is equal, and movements about as large (seemingly not as large because a greater mass of flesh is lifted on the healthy side); every muscle of the cheek and lip on the left side is present and normally active. To galvanism the reactions in the nerve and in the muscles with from 8 to 9 elements of Stöhrer's battery are normal: KaC>AnC; equal on the two sides.

Vaso-motor phenomena: With the exception of the white spot mentioned above there is no difference between the two cheeks as regards color; when the patient blushes the two sides of the face and head become equally red; and nitrite of amyl affects them similarly. The patient and her mother have observed that when she gets warm no perspiration appears on the wasted cheek. I have made one or two observations upon the temperature of the two cheeks, but have found no marked differences.

The treatment thus far, for about two months, has consisted in tri-weekly galvanization of the affected cheek, the kathode on the wasted cheek and the anode on the nape of the neck;

the current of 8 or 10 elements of Stöhrer's battery being used. No noteworthy result has followed.

To my knowledge, only four other instances of this rare and obscure disease have been observed in this country: one each by Dr. W. A. Hammond, by Dr. W. H. Draper, by Dr. Bannister, of Chicago, by Dr. Beverly Robinson, of New York. In the excellent article by Eulenburg, in vol. XIV, of *Ziemssen's Cyclopedia of Medicine*, only sixteen well authenticated cases are reported.

I have, during the past two years, seen Dr. Draper's patient repeatedly, and can say that the disease in her case has decidedly advanced about the temporal and pre-auricular regions. Changes seem also to be going on in the lower jaw, as the teeth have rapidly decayed, and intense neuralgia has occurred. I should add that in this patient I have at all times been able to demonstrate the integrity of the electro-muscular and electro-neural reactions, and of sensibility.

At the November meeting, Dr. E. C. Spitzka read a paper on the "Merits, motives and prospects of the present movement for Asylum reform,"* and Dr. W. A. Hammond read a brief paper on "Cerebral symptoms from impacted cerumen in the ear." The paper consisted essentially of a number of cases in which marked cerebral symptoms were developed, such as hallucinations, temptations to suicide, tinnitus aurium, noises, voices, etc., and disappeared upon the removal of impacted cerumen from the ear.

Commenting upon the cases, the doctor gave his view with reference to the cause of the roaring produced by the introduction of the end of the finger into the ear—namely—that it was the circulation in the finger that was heard, the finger simply acting as a conductor of the sound; adding further in explanation of his theory, that if a cork be introduced into the ear and the hand removed, no sound would be heard, but that if the hand were again brought in contact with the cork the roaring would recommence.

Dr. C. A. Leale referred to two cases which illustrated the practical importance of the subject of the paper. One was that of a young man, who, on account of the gradual development of cerebral symptoms, was advised to discontinue his college course. He suffered considerably from pain in the ears, and in obtaining a history of the case it was learned that when two years old, fourteen years previously, he had been in the habit of putting beans into his ears. One bean had been removed from one ear, but another remained, became surrounded by wax, pressed upon the tympanum, and excited all the cerebral symptoms recognized. The bean was removed, and the symptoms, to a very great extent, disappeared, but the prospects of the young man were ruined, for he had already given up his studies and engaged in some simple farm labor. The second was that of a lawyer, fifty-four years of age, who had been recommended to give up his profession because it was almost impossible for him to hear the judge's remarks or the answers from a witness. On examining the ears nothing at first could be seen. The external meatus was small, and the canal could be illuminated only with considerable difficulty. Finally, a dark mass was discovered, which was readily removed with warm water injections, and it proved to be inspissated cerumen. With its removal all disagreeable symptoms disappeared, and the gentleman renewed his professional labor.

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Dr. Gray remarked, with reference to the hallucinations which had been attributed to impacted cerumen in the ear, that the question arose whether in any of the cases the hallucination would have been present had there not been a predisposition to insanity, either hereditary or from the previous existence of insanity from other causes. With an excitable condition of the nerve centres, produced by previous insanity, it was easy to understand how any peripheral cause might develop the predisposition.

Dr. Hammond remarked that he was not able to state whether, in the cases reported, there was any predisposition to insanity. The curious part of some of the cases was, that the patients did not suffer from illusions, but rather from what might be called perversion of hallucination. They did have noises in the ears, and those noises were converted into real voices. He did not know of any example in the entire range of cerebral phenomena, in which a perverted sensation was converted into speech for instance, or anything which conveyed a distinct idea to the senses. The doctor then referred to the danger which attended the introduction of sea-water into the ears, and mentioned cases in which marked cerebral symptoms had been developed in consequence of such irritation.

Dr. Webster regarded the statement made by Dr. Hammond with reference to the cause of the noise in the ear when the finger was introduced into the external auditory meatus, as entirely new, and one which he could not accept. He believed that the noise was produced by pressure of the air against the tympanum, and not simply by introducing the finger into the ear.

Dr. Kiernan remarked, with reference to the hallucinations based upon perverted sensation, that a patient came under his observation who believed that he had a doctor in his abdomen who was constantly talking to him, but the speaking was not through the ears. With reference to inspissated cerumen, he had found many insane patients whose ears were filled with it, but the removal of the wax had not in any case been followed by removal of the hallucinations.

Dr. Roosa remarked that after spending some years in the examination of cases, he had not been able to satisfy himself with reference to the etiology of inspissated cerumen. With reference to its existence he would simply say, that he did not believe any person who had had inspissated cerumen more than once in his life had sound ears. If inspissated cerumen occurred more than once in the same person it was evidence that something of the hearing power had been lost, and that it would become still further impaired. With reference to hallucinations, Meyer, of Hamburg, had removed inspissated cerumen from the ears of a large number of insane persons, but only in two cases did the hallucinations disappear after the removal of the wax.

Dr. Sessell had directed his attention to one case in which hallucinations were removed by the removal of impacted cerumen. With reference to the causation of noise in the ear when the finger was introduced into the auditory canal, he believed the chief cause was the pressure exerted by the finger, and that the arterial circulation had but little to do with it.

Dr. Shaw believed with Dr. Gray, that mental symptoms would be produced by impacted cerumen only in those who had a predisposition to mental disease.

Dr. Leale made special reference to the irritation produced by crystals from sea-water left in the ears after bathing, and the importance of carefully washing the ears out with warm water.

ABSCESS OF THE BRAIN.

Dr. J. C. Shaw presented specimens from a case of abscess of the brain.

The patient was a man thirty-seven years old, who had had suppurative inflammation of the ear for a number of years. Two weeks previous to his death he had a little extra pain in the ear, but it passed with slight notice. The pain becoming more severe about four days before his death, a consultation was held, and an incision was made over the mastoid process for the relief of what was supposed to be inflammation affecting the mastoid cells. The man, however, became rapidly worse, and passed into a state of coma. The mastoid process was also trephined, but no relief was obtained. At autopsy, a small necrosed point was found in the dura mater just above the ear, with a sinus leading up to an abscess. There was extensive meningitis, which was regarded as the probable cause of death. The case was mentioned for the purpose of drawing attention to what was believed to be

A NEW FACT IN CONNECTION WITH MENINGITIS AND DISEASE OF THE MIDDLE EAR.

Dr. Shaw stated that he had occasion to make post-mortem examinations in five or six cases of meningitis associated with middle-ear trouble, and the question arose, how was the meningitis produced?

In three cases he had been able to trace the pus along the Gasserian ganglion and to the fifth nerve.

Dr. De Rossett thought we should not forget, in this connection, the possible transference of trophic changes from one portion of the body to another.

Reviews and Bibliographical Notices.

I.—DURET: CEREBRAL TRAUMATISMS, &c.

ETUDES EXPERIMENTALES ET CLINIQUES SUR LES TRAUMATISMES CEREBRAUX. Par le Dr. H. Duret. Pp. 469. (*Experimental and clinical studies on cerebral traumatisms, &c.*)

This is an admirable work in many respects, and is a positive acquisition in the important and difficult department of physiology and medicine to which its statements relate. After a few judicious remarks in the way of an introduction, a short chapter is given to definitions and to a statement of the scope of the work. We cannot do better in beginning, than to extract portions of this first chapter. Says the author: "The nervous accidents caused by mechanical injuries of the brain may be distinguished as *primitive*, *secondary* and *tertiary*. By *primitive* accidents, we mean those which begin at the place and moment of the wound, or in a very short period afterwards, such as the complex pathological states, to which the names cerebral *commotion*, *compression* and *contusion* have been given.

"*Secondary* accidents are those which have their point of departure in the inflammatory reaction excited by the mechanical lesions in the midst of the nervous centres, produced by outside causes. They do not usually appear before the second or third day. Under this head we would include *meningitis*, *encephalitis*, *cerebral abscess*, *&c.*

"But when these phenomena have subsided or disappeared, and when the patient may even appear to have more or less completely recovered, often after months or even years have elapsed, certain disorders may manifest themselves, having their origin in the pathological residua of the traumatism and its reaction, and such are the *tertiary* accidents of cerebral traumatisms.

"As regards motion, the phenomena consist in local paralyses, hemiplegias, monoplegias of the face, eyes or members, and in certain 'contractures,' of atrophies (muscular) by descending degeneration, or even in epileptiform attacks. For sensibility, the disorders are chiefly 'localized anæsthesias, hyperæsthesias, neuralgias, &c., &c. In the sphere of mental action, various disorders appear, such as delirium, localized or generalized, general paralysis of the insane, monomania, suicidal insanity, loss of memory, of language, &c.

"In the first part of this work we shall be occupied in considering the pathological physiology of the primitive accidents, such as cerebral commotion, compression and contusion. If we do not mistake ourselves, great practical confusion prevails as regards the signification of these terms. We do not know as yet the exact limits of the manifestations which belong to each of these pathological entities. In the presence of a patient suffering from a cerebral traumatism, it is not rare to see an instructed and conscientious surgeon hesitate between the diagnosis of compression and contusion, or ask himself if he is not concerned with the effects of a commotion, and even after the death of the patient, the hesitation may not cease. In effect, these pathological states are superposed the one on the other, and hence complicate the case to the observer. Each of the above denominations corresponds almost always to multiple lesions, which may occupy parts of the brain the most diverse, either the hemispheres, medulla, or the cord, or the vascular or membranous envelopes of these parts.

"It seems to us, that to throw any light on this pathological complex, it is necessary to ascertain what manifestations appreciable in pathology, lesions of special parts could give rise to. We have accordingly proposed to ourselves the following questions: What is the rôle of lesions of different parts of the hemispheres, of the medulla and cord, of the dura mater and its nerves, of the cerebro-spinal liquid in the production of the primitive accidents of cerebral traumatisms?

"This analysis once made, we shall proceed to apply the results to the study of the pathological entities: commotion, compression, contusion. This is without doubt, a long and difficult work, but we have not been deterred from undertaking it. In treating such a subject, lucidity in conception, and method in exposition are equally indispensable. This is why we have indicated didactically the course we have proposed to follow. In the first we shall study successively from the point of view of the traumatism: 1st. The cerebro-spinal shock. 2d. The effects of an excess of intra-cranial pressure. 3d. The rôle of lesions of the dura mater. 4th. The rôle of vascular disorders. 5th. The rôle of lesions of the cerebral cortex and of other nervous centres, medulla, cord, &c. To this end we shall employ almost exclusively experimental researches. Experiment alone permits of clear analysis, since clinical cases are generally quite complex. In the second part we shall apply the results obtained in the explication of cerebral commotion, compression and contusion as they are conceived by surgeons to-day. Then will follow the synthesis rendered more easy by the light of experimental physiology." (Pp. 1–3.)

In the above extract is to be found the plan of this suggestive work on a subject attracting very properly a considerable measure of attention from physicians, surgeons and medical jurists, from the latter especially, in this age of railway accidents and the like, in which, with painful frequency, there are injuries, direct or indirect, of the cord, or brain, or both, of such a nature and under such circumstances as to give occasion to troublesome litigations for the recovery of damages in case of personal injuries. But we will now pass on to give the reader an abstract of the results, with remarks on their value and on the adequacy of the methods employed in reaching them.

Shock is defined in general as follows: "The expression 'shock,' in physiology designates a phenomenon, sudden in its appearance, produced instantaneously by an external cause. This phenomenon consists generally in a sort of enfeeblement or vanishing of the functional activity of the organ attacked. *According to our view, the morbid action is not produced, only by the intermediation of the sensory nerves.* We have employed the words *cephalo-rachidian shock*, to indicate the arrest or sudden suppression of encephalic function, coming on in consequence of a blow on the cranium, and is produced by the intermediation of the cerebro-spinal fluid, transmitting the 'vulnerant' action to certain regions of the brain, capable of engendering all the phenomena observed." Again, "when we come to a study of the vascular disorders which occur in the brain, as consequences of traumatisms of that organ, we shall expose the important rôle of the different kinds of *vascular shock:* by sudden pressure (*vascular*) and the *reflex* vascular shock. The *embolic* and *hemorrhagic* varieties of encephalic shock are to be taken account of, and explained and compared with *cephalo-rachidian* shock. They constitute what may be called *medical* shock, while the former varieties (cephalo-rachidian) may be called *surgical*.

"There exists, finally, a *nervous shock*, properly speaking, in which no liquid intervenes. It is when a sensitive nerve (injured), by an action of arrest suddenly diminishes or suspends the action of the brain or higher nervous centres. Such is sometimes the action of the brain on the cord, of the pneumogastric nerve on the heart. The *cephalo-rachidian* shock, which is the principal object of our studies, has never been, we believe, indicated and explained until the present." (Pp. 4, 5.)

Dr. Duret denies that the multiform and grave phenomena of shock, as he describes it, can be produced in the way ordinarily supposed, viz.: that of a hypothetical molecular disturbance, not often manifest to the microscope, and resulting from external violence to the cranium. He gives pretty good reasons for the positions he assumes. Neither can we find an explanation of the phenomena in question by a rational appeal to even the most recent physiological notions concerning the rôle of the hemispheres. In the lower animals, the hemispheres may be contused, lacerated, or even wholly removed, without producing the phenomena of shock. To a certain extent, the same statement applies to man. But the symptoms of shock are often manifested when there are very inconsiderable lesions of the brain, or even none at all to be found after death. Then how shall we explain cerebral shock? Its most common and characteristic phenomena are *sudden partial arrest of respiration, and of the heart's action, followed by disturbance*, or even *loss of consciousness.* Dr. Duret would refer these phenomena to an injury of the medulla rather than the brain. In it lie the cardiac and respiratory centres. The exact mechanism of these consequences is explained farther on in the work. But how explain the disturbed mental action? The author would explain it on the one hand, by postulating lesions of the nucleus of the hypoglossus, and of other of the motor cranial nerves, in such way as to obscure or prevent the *expression* of the workings of the mind, and on the other hand, by cutting off the sensory tracts in the medulla, which convey impressions from the outer world to the cerebral cortex. And so between the cessation of mental action, which it is *assumed* would result from cutting of its communication with the outer world, and the impossibility of expressing either thought or emotion, by reason of a paralysis of the nervous apparatuses involved in their expression, the individual is either actually unconscious, or at all events *appears* to be so to the outward observer. In the support of these views, Dr. Duret enters into certain discussions, into which it is our purpose briefly to follow him, all the more so, as we believe them to be in great measure erroneous.

"What is Life?" says he. The reply is as follows: "It is in the animal the reaction of the nervous centres under influences which are being or have been transmitted to them by the sensitive nerves, sensorial or vegetative."

In stating the relations of mental action and development to the exterior nervous apparatuses and external world beyond them, he remarks: "In one word, *we owe to the external world* the life *intellectual and voluntary, that is to say, the life of relation; and the vegetative life does not appear and is not developed except by excitations from centripetal conductors.* Hence, it is easy to understand that if these two orders of relations with the external world are abolished by the destruction of sensitive fibres, that life is extinguished." If these assumptions were anything more than partly true, we might expect *a priori*, that the central nervous apparatuses to which sensory nerves go, especially in the higher parts of the nervous system, would be necessarily in a very imperfect state at birth, or until exposed to sensorial excitations from the outer world. This view, which is by no means novel or uncommon, is the one adopted by Dr. Duret. He refers to his own valuable researches (*Archives de Physiologie Norm. et Path.*, 1874 and 1875,) on the circulation of the brain and on the cerebral functions, to show that the structure of the brain is so imperfect as to strongly support his views concerning the absolute dependence of the brain and higher parts of the central nervous system for its existence and development on excitations from the external world by way of the sensory nerves. Sander, Soltmann, Rouget and Munk are quoted in support of his position. But we can go no farther than to say that we thoroughly disagree with the author in respect to the validity of the assumptions at which we have glanced. His statements are tissues of error and truth, not even skillfully combined. They do not have even the merit of novelty. His definition of life is partial and faulty, as it would be easy to show. His dictum as it stands, as to the absolute dependence of the central nervous system for its appearance and development, is contradicted by facts at all points. Take for example the chick in the act of escaping from its shell, with its head just out, and in a few minutes opening its eyes and *seeing* an insect on a blade of grass within its reach, and picking it off with precision and swallowing it; or take the young serpent, as we have seen, let out of its egg-case by cutting it open with a pocket-knife, and which, when liberated, immediately ran and hid itself beneath a clod. But not to multiply cases even among the higher animals, we would refer to the papers just published of Tarchanoff,* in the last two numbers of the *Revue Mensuelle*, and in which he shows that in many animals, even the cortical motor centres are developed before birth, as was proved by examinations, anatomical and physiological, of young animals, both immediately after birth *and before it.* In such cases the central nervous apparatuses, which, according to others as well as M. Duret, can only be developed after the nerves leading to them have been the seats of excitation in their peripheral ends, are found to the contrary to have been developed in the absence of this prime condition.

Then again, in explaining how it comes to pass that affections of the medulla disturb the mental functions, the author does not seem to us to have arrived at a just conception of their *modus agendi.* But we may find occasion to return to this topic a little later.

Dr. Duret began a series of experiments on animals, especially rabbits, which consisted partly in the injection with some force into the cranial cavity through a small hole, on the *surface* of the brain, certain quantities of water, or other liquids, after which the animal immediately died. The post-mortem showed rupture of the brain from the great ventricular cavity *outward* through the substance of the hemispheres, and also distension of the Sylvian aqueduct and medulla. Upon considering these cases, especially those unexpected lesions, the author says: "A luminous idea suddenly rose in my mind. This distension of the medulla, this rupture from within outwards, this dilatation of the aqueduct of Sylvius and of the central canal, had been produced by the enormous tension of the *cephalo-rachidian* fluid. Under the influence of sudden and severe pressure exerted on the surface of the hemispheres, the very large quantity of the cerebro-spinal

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J. de Tarchanoff: "Sur las Centres Psycho-Moteur des Animaux Nouveau né et Leur Developpement dans Differentes Conditions," *Revue Mensuelle*, October and November, 1878.

fluid contained in the lateral ventricles had been rapidly driven out, being made to traverse under pressure, the aqueduct of Sylvius into the fourth ventricle. The aqueduct of Sylvius was dilated and torn. The fourth ventricle receiving *suddenly* an enormous quantity of fluid, which could not find easy escape by the central canal, or by the foramen of Magendie, the fourth ventricle had become to such a degree distended as to burst. At the same time there was dilatation of the central canal, of the spinal cord, and of Magendie's cavity." (P. 11.)

According to the opinion of Dr. Duret, this mode of accounting for some of the immediate consequences of cerebral traumatisms was one of the most important discoveries made in the course of his researches. In order to avoid some actual or possible objections to his experiments and the deductions from them, they were varied in many respects, until it appears that we may accept both with a good degree of trust. The same kind of gross lesions were often found to result from sudden blows on the head without the injection of fluid into the cranial cavity. By them Dr. Duret would explain in many cases sudden death following blows on the head, and many other grave conditions so resulting, which are not at once or at all fatal. And this is briefly the *cephalo-rachidian shock* of our author, and on which he very properly lays much stress.

In order to render his dicussion intelligible, Dr. Duret gives a very clear *resumé* of researches, up to date, regarding the system of lymph-spaces and channels about and within the brain. Beginning with the larger subarachnoidal spaces beneath and about the brain, filled with fluid and called "lakes" (*lac* sylvien—*lac* cérébelleux inferieur—*lac* cérébelleux superieur), these spaces are traced outwards along the larger fissures (which centre at the "lakes"), such as the *Sylvian* fissure, and which are called *flumina*, and these to smaller fissures branching from the larger ones and called *rivi*, and from these again to still other and smaller fissures, branching from the *rivi*, called *rivuli.* From these finally, there are small branching tunnels, so to speak, in the nervous substance, which the arteries enter and follow to their final terminations in the capillaries. The vessels do not fill these tunnels in the brain substance, the space they do not fill being occupied loosely by delicate connective tissue, the meshes of which are filled by the "cephalo-rachidian" fluid, which distends not only the spaces about all the vessels in the brain substance, but in a regressive order also the "rivuli," the "rivi," the "fluminæ," converging thus from every direction on the central reservoirs of the "cephalo-rachidian" liquid, in the "lakes" or subarachnoidal spaces beneath the brain. These same lymph-spaces may be traced around and into the medulla and cord, and along the sheaths of the nerves, especially the cranial, and among them, most evidently along within the sheaths of the olfactory, auditory and optic nerves, even in one case to the perilymph in the labyrinth, and in another to the liquid which fills the space within the globe of the eye, in front of the lens. The same spaces filled by the same liquid may even extend along the spinal nerves, within their sheaths, to their peripheral ends.

We thus have a most comprehensive and penetrating system of lymph-spaces, extending from the great "lakes" or subarachnoidal spaces beneath the brain, in one direction down the cord and peripheral nerves, cranial and spinal, and in the other, a system of branching lymph-channels, penetrating the brain substance by successive divisions, until they reach in microscopic fineness the ultimate fibres and cells of the brain. Cerebro-spinal fluid (lymph) and delicate connective tissue webs, go hand in hand through a complex system. Our knowledge of this system of lymph channels has mainly resulted from the researches of Robin, His, Schwalbe, Axel Key and Retzius, Golgi, Ranvier and many others, including in the list the author of the present work.

Dr. Duret speaks of the use of the cephalo-rachidian liquid as follows: "Its principal physiological use, is to preserve the nervous elements against cardiac shock, or the excess of vascular tension, not only of the elements in the nervous centres, but even in the nerves and nervous parts of the organs of sense. If the tension of the blood is augmented a more considerable quantity of the liquid is exuded, and if the subarachnoidal spaces do not deplete the lymph channels, cerebral œdema results, as may be observed in certain cases of inflammatory reactions provoked by traumatisms. The tension of this liquid exceeds the pressure of the atmosphere, as has been proved by decisive experiment. It plays, according to our author, an important part in cerebral shock. He says: "Can a displacement of parts (of the brain and medulla) be produced under the influence of shock? The answer to this question is affirmative, if the cranium is elastic, and if the cerebro-spinal fluid has a way for momentary escape from the cavities it occupies." In case of a shock, especially, received on certain parts of the cranium, there always occurs "a sudden distribution of the shock or gross impulse to all those regions to which the cerebro-spinal liquid circulates. Under the influence of the sudden pressure, the liquid is expelled from the 'lakes' into the 'fluminæ,' from these into the 'rivi,' and from these again into the 'rivuli,' and finally" the impulses are propagated along the liquid which fills the lymph-spaces about the vessels, onwards to their final ramifications amid the nervous elements. "Thus we may explain the thousands of small vasal ruptures permitting small hemorrhages, which as consequences of shock, are strewn in the nervous substance, in the most divers places." To this it seems to us the author may have added, that there are also under the circumstances given, delicate and wide-spreading ruptures of the nervous elements one from another, independently of the hemorrhages, as well as caused by them. The physical conditions of the case render such a deduction almost a necessary one. The author does not overlook this phase of the subject. He has not, however, insisted on it, as it deserves.

Starting with the fact, that the cranium is elastic, and temporarily changes its form when struck, he recites the results of a number of experiments on crania filled with paraffine, instead of brain substance, and they are of considerable interest to those who desire to study intelligently the physics of localized mechanical violence, done to the cranium and its contents. But we cannot go into this subject at present. And we have given so fully the views of Dr. Duret on the subjects just mentioned, because they are of great value to the physician, surgeon and medical jurist. They afford us a rational statement of the nature and results of "cerebral shock" resulting from traumatisms.

After this more general part of the work, Dr. Duret begins a detailed study, into which we may profitably follow him. In the first place a "physiological explanation of the lesions produced by the cephalo-rachidian shock," is given. Although it will in some measure lead us over the same ground that we have already traversed, yet we shall find much of interest to our readers who may not have seen the work itself. The first generalization made, is that "the lesions produced by the shock, *occupy principally the spaces where the cerebro-spinal fluid circulates.*" These lesions are divided into two classes, "superficial" and "interstitial." Among lesions of the first of these classes, those of the convexity of the hemispheres are first described. In case of a blow on the cranium, the resulting lesion (superficial) "may occupy the point percussed, or the opposite extremity of the axis of percussion, that is to say, corresponding either of the *cone of depression* [cône de depression] or *cone of elevation, or projection* [cône de soulevement]." The former is the depression of the elastic cranial wall, at the point where the blow is struck, the latter being the sudden *projection* of the same cranial wall, at the side opposite to the one occupied by the "cone of depression." The lesions at the "cone of depression," where the cranial wall is driven in on the brain substance, are mostly of a gross surgical character. But not so those which occur at the "cone of projection," which is carried away from the brain substance, as it were. In explaining these latter lesions, Dr. Duret says: "We do not admit the doctrine of lesions by *contre-coup*, as it is formulated by classic authors." He explains these lesions as follows: "The lesions observed at the remote extremity of the axis of percussion, opposite to the point percussed, are the result of the action of the "cone of projection." It creates suddenly a void, and aqueous and sanguine liquids are suddenly driven into it to fill it, and then result ruptures of vessels, and blood phlyctænulæ *beneath the pia mater*. That which goes to confirm our opinion, is a study of the appearance of these lesions. They occupy an oval-shaped space, sometimes circular or elliptical, corresponding to the base of the *cone of projection*, and their maximum is at the centre of this base." (P. 38.) These lesions are then described and figured in various plates at the end of the volume. But we cannot now enter farther into their consideration.

The mode of occurrence of "lesions of the hemispheres at their base," is described as follows: "Lesions of the base of the hemispheres are very frequent and very pronounced, because there we find the 'aqueous lakes'—the *Sylvian*, which in blows on the vertex, support the momentum of the shock. In effect, in a stroke on the head, on the most convex part of the head, all the vertex constitutes the cone of depression. At the opposite extremity of the axis of percussion, there is in this case no cone of projection, on account of the firmness of the base of the cranium. The cerebro-spinal fluid, being driven from the convexity flows towards the 'lakes' at the base of the brain, and produces a sudden inundation of surrounding territories. The peripeduncular canals are far from being sufficient to accommodate so violent an irruption of fluid. The small vessels which traverse the 'lakes' are ruptured, the neighboring parts are inundated by blood, and at times the nervous substance is involved, and its structure destroyed by the violence of the flood. In certain cases, the visceral layer of the arachnoid is lacerated, and blood mixed with cerebro-spinal fluid fills the cavity of the arachnoid, either ascending in the same towards the convexity of the hemispheres, or what is more frequent, descends between the two layers of the arachnoid, around the peduncles, pons and medulla. It is remarkable to observe how faithfully the hemorrhages occur in, and outline the scheme of the 'lakes,' as already described. In the dog [killed by a stroke on the head] for example, the *Sylvian* and *central* 'lakes' and the peripeduncular canals, are already outlined by the hemorrhages, and on the confines of these lymph-spaces may be seen the localized hemorrhages, radiating along the 'fluminæ,' and beyond to variable distances, and at times penetrating the nervous substance." (Pp. 39, 40.)

Similar vascular lesions and frequently ruptures of nerve trunks, or of the nervous substance of the peduncles, pons and medulla also occur. The spinal cord, even down to the lumbar region, does not escape these vascular lesions.

Dr. Duret next gives detailed attention to lesions of the ventricular cavities resulting from blows on the head. He says: "One general fact must be made prominent in the outset, namely, that in case of a blow on the head, a *wave of percussion* is produced in the ventricular cavities. * * * The ventricular wave produced by the percussion is always more decided if the violence is exerted on the forehead or vertex. Under such circumstances a very voluminous *cone of depression* is formed, especially if the percussing body has a large surface. The ventricular cavities are then suddenly effaced, and the wave of liquid (ventricular) engages with all the more violence, since a cranial *cone of projection* is not possible. The liquid traverses the aqueduct of Sylvius, and is hence violently driven into the fourth ventricle. From this it passes by the foramen of Magendie, which is torn if not very large, and rushes farther on into the posterior cerebellar 'lake,' and under the medullary and spinal pia mater. The lesions of the medulla, in the commotion, occupy principally the posterior parts of the lateral ventricles, the aqueduct of Sylvius, that part of the medulla which corresponds to the fourth ventricle, especially its inferior parts, the foramen of Magendie is ruptured, and the central canal of the spinal cord receives part of the wave of fluid, which often results in violence. Clots of blood fill more or less the ventricular cavities in the walls of the same and in the floor of the fourth ventricle, especially in company with lesions of the nervous substance in the lower part of the floor of this ventricle. At the moment of the violence, two liquid waves are started, the one external to the brain (peripherique), the other from the interior (cavitaire), which both pass towards the foramen magnum, and which converge at that point to produce the principal lesions, caused by violent movements of the cerebro-spinal liquid, as might be expected *a priori.* These lesions are described at length by Dr. Duret, and duly illustrated by colored drawings at the end of the volume. But we will postpone the remainder of our notice of this interesting work to the next issue of the Journal, in which other and even more interesting parts will be fully noticed on account of their practical importance, not only for the physician, but also for the surgeon and medical jurist.

(*To be continued.*)

II.—THE MEDULLA OBLONGATA.

I. KRANKHEITEN DES VERLAENGERTEN MARKS. Pp. 831–960. *Handbuch der Krankheiten des Nervensystems*, Bd. II., zweite Haelfte. Von Dr. Wilhelm Erb. Zweite Auflage, 1878. (*Diseases of the Medulla Oblongata, &c.*)

II. MOELLE ALLONGÉE OU BULBE RACHIDIEN. *Dict. Encyclopedique des Sci. Med.*, deuxieme serie, tome huitieme. Premiere partie. Pp. 301–328. Du meme, deuxieme partie. Pp. 604–788. (*The Medulla Oblongata, or Rachidian Bulb.*)

No part of the central nervous system has more seriously challenged the attention of the anatomist and physiologist, or more thoroughly baffled both, than the medulla and the pons, with which latter the medulla sustains such close relations. So unfruitful do the elaborate anatomical investigations of this part of the cerebro-spinal axis appear to some judicious observers, as to lead one of the foremost among them to declare during the past year, in the important work cited at the head of this notice, that "the anatomy of the medulla oblongata is yet in many and perhaps the most important relations, imperfectly or not at all known. "The relations are extraordinarily difficult to unravel. The connections of the structures of the medulla oblongata with the recognized structures of the spinal cord, and with the sections of the nervous system lying above it on the other hand, are on the whole extraordinarily complex, manifold, intricate, and in spite of the most extreme care in anatomical investigation, are yet in great measure in darkness. It is hence easily enough to be explained, that the views of the best observers, concerning the minute structure of the medulla oblongata, are yet in many points widely asunder." So much for the present state of the anatomy of the medulla. As regards the *physiology* of the medulla, the same conservative and cautious observer (Erb), speaks as follows: "Not less imperfect and doubtful than the anatomy of the medulla oblongata is its physiology. As regards the more intimate and important questions of its physiology there has not been obtained as yet any indisputable knowledge." And such in most instances is the sum of the testimony of the best observers.

And yet perhaps, not another part of the central nervous system has been the object of more elaborate investigations.

It has been most carefully studied by such anatomists as Koelliker, Stilling (especially the latter), Clarke, Schroeder Van der Kolk, Deiters, Meynert, Luys, Dean, Henle, Sappey and Duval, Gerlach, Huguenin, Stieda, Faraboeuf, Flechsig and a host of others. And yet after all, we have the judgment above uttered by Dr. Erb, from which it would appear that but little substantial progress has been made.

This is unhappily too true. But it seems to us the statements quoted above underrate our real knowledge of this part of the nervous system. A comprehensive survey of our present knowledge of the anatomy, physiology, and pathology of the medulla, we believe will warrant us in going farther than does Dr. Erb. It is our purpose in this article to briefly state what we believe to be established, and what probable in regard to the structure and functions of the medulla.

It may be laid down as a cardinal principle for guidance in a study of the nervous system, that our knowledge of its structure and functions, derived from a study of its simpler parts, is to be applied industriously in the study and interpretation of more complex parts. These latter parts differ chiefly from the former in their degrees of complexity, rather than in *kind* of structure and function. In applying this simple and well known rule in a study of the medulla, we should rise from a study of the spinal cord, which is simpler and better understood, carrying up the results to the medulla, which, whatever else it may have or do besides, contains the same anatomical elements and discharges similar functions to the cord. The sensory or esthesodic, and the motor or kinesodic tracts, both grey and white of the cord, are continued upward through the medulla, though changed in their form and relations. To these many new parts or what seem to be such, are added, by which at least its size is augmented. As compared with the cord the medulla is considerably changed in form, as already said, partly by changes of relative position of masses of matter, and partly by special additions of grey matter and fibres, which increase its size but which are peculiar to the medulla in appearance rather than in fact. The same principles of interpretation of structure and function which apply to the cord apply to the medulla. It presents but little if anything radically new as compared with the cord below it. But it is more complex. If these remarks are true, it is necessary for us to state in the outset of a study of the medulla, what are the more important of our items of knowledge of the structure and functions of the spinal cord. This we shall accordingly do.

There is one point in the nervous system only in which there is seemingly a complete *anatomical* separation of its sensory and motor tracts, viz.: just before the nerves plunge into the cord and medulla. Ever since their discovery by Sir Charles Bell, it has been known of the two roots of the mixed nerves, that the posterior is sensory and the anterior motor. Outwardly from this point, the fibres of these two bundles as a rule mix, and yet are known to preserve their characteristics and to go to their destinations in the muscles, glands, &c., and to the sensory surfaces, as certainly as if they had remained distinct in space relations in their whole course, as they are before entering the cord. The esthesodic and kinesodic tracts are distinct, and known to be so, in spite of appearances, from the separated roots of the spinal nerves, outward to the periphery of the nervous system. On the other hand, after the disappearance of the bundles of fibres composing the sensory root into the posterior aspect of the spinal cord, and of those composing the anterior or motor root, into the anterior aspect of the cord, we lose sight of them, and the most penetrating anatomical investigation has not been able thus far to follow them to any considerable distance, so as to tell by such means what course they actually take in the cord, in any considerable numbers. And so far as we can judge from present appearances, it will always be difficult if not impossible to follow anatomically, the fibres of the cord to any considerable distance, unless by indirect methods, such as those adopted by Flechsig in carrying out his remarkable researches. But this much may be said with considerable certainty, that every advance in our knowledge goes to render more probable the assumption, that however intimate may be the relations of the motor and sensory tracts in the cord for reflex purposes, they are yet not less distinct in other respects than are these two tracts in the roots of the spinal nerves. They pursue a parallel but a definite and distinct course upwards into the brain, in all probability. This general statement being admitted, as we think it must be, we are prepared for further general statements as regards the cord as a whole. There is every reason to suppose that the fibres of the motor and sensory roots of the nerves when they enter the cord, terminate sooner or later wholly in its grey matter. There is no good reason whatever for supposing that any fibres from either root passes without interruption up to the brain. They all probably enter the grey matter of the cord.

Confining our attention, now, to the fibres of the posterior or sensory root, and admitting that they enter and terminate directly or indirectly in the cells of the grey matter, we may go farther and state that they do so apparently for two general purposes, and two only, viz.: for immediate reflex purposes, and that sense impressions may be conveyed upwards to the brain, where they may be manifested in consciousness. There must then quite plainly be, two classes of mechanisms in the cord to subserve these purposes, however powerless the anatomist may be to make a clear demonstration of them. One is to convey sense impressions entering the cord to corresponding motor mechanisms on the same level, and in the same side of the cord, or to a higher level in the same for reflex purposes, the other to convey impressions up to the brain.

To convey nervous impulses upwards toward the brain, there must be some provision, such as is known to exist in other parts of the nervous system for similar purposes. This is usually nerve fibres, and there is every reason for believing that this is true for the spinal cord. The *routes of preference* for such impressions, are, in all probability, chiefly in the bands of fibres in the lateral columns of the cord. Yet it is necessary to admit that such *may* be conveyed upwards in its grey matter, in most if not all its parts. That sensory fibres emerge from each segment (corresponding to pairs of nerves) of the grey matter of the cord and medulla, and that these pass upwards to terminate in the grey matter of the basal system of ganglia, or pass through the internal capsule upwards to the grey matter of the cortex of the brain proper, there can now be little queston. These fibres may, some of them, pass upwards in the posterior columns as Schiff supposes (though we think this is doubtful), but that they pass mostly in the mass of the lateral columns, seems tolerably certain. That they take their origin in the grey matter of one side of the cord, cross the middle line, and ascend toward the brain in the opposite half of the cord, seems highly probable.

Then, again, it is pretty well established that fibres arise from the cells of the grey matter of the cortex of the brain (motor zone), and that from thence they pass downwards to the level of the basal system where some of them terminate, while others pass by the basal system through the internal capsule to terminate,after crossing chiefly over the middle line, in the grey matter of the medulla and cord, so as to connect by this means various horizons of the grey matter of the cord and medulla, with particular regions of the cortex, while other fibres connect the grey matter of the basal system with various horizons of the grey matter of the medulla and cord, for automatic rather than voluntary purposes. That these fibres of both kinds (motor) are contained chiefly in the lateral columns of the cord, and probably also in the columns of Tuerck of the anterior columns, and that they terminate in the cells of the kinesodic tract, so as to excite the motor apparatuses, from which the motor roots of the spinal and medullary (cranial) nerves arise, seems quite probable from every point of view.

We thus have in the cord, provision for reflex action and for the transmission upwards towards the brain of sensory impulses, and on the contrary downward from the brain of motor impulses, and we seem now in position to lay down what are the mechanisms for such actions, and to no inconsiderable extent, their topographical relations in the cord. The anterior horns of grey matter are undoubtedly motor, while the posterior ones are no doubt chiefly sensory.

Now, the point in all this is, that what is true for the cord is also true for the medulla. If we have any certain knowledge of the former, to that extent we have a certain knowledge of the latter. The same tracts of matter which exist in the cord are found continued up into and through the medulla, to the grey matter of the ganglia of the basal system and cortex of the brain, including no doubt that of the cerebellum.

It is now known with a fair degree of certainty that the anterior pyramids of the medulla are largely composed of bands of fibres derived from the lateral column of the opposite half of the cord (crossed pyramidal tract), and from the anterior column of the cord of the same side (column of Tuerck—direct pyramidal tract). It is also known that the fundamental tracts of the anterior columns of the cord, as they approach the medulla, are pushed outwards so as to pass beneath, and then outward from and behind the olivary bodies, and then ascending behind the pyramids they approach the raphe, decussate probably and ascend toward the opto-striate body. It is known also that the lateral columns of the cord, as they pass upwards to the level of the medulla, are probably disposed of as follows: A tract of motor fibres, already mentioned, which crosses over from one side to the other at the pyramidal crossing, so as to join the opposite anterior pyramid of the medulla. A tract of sensory fibres which enters the lateral part of the medulla and bends toward the raphe and ascends through the tegmentum of the crus, partly to the cells of the optic thalami, and partly through the posterior part of the inner capsule to the cortex of the hemispheres. That another tract of the lateral columns, embraced in its posterior third, passes upwards, enters the corresponding restiform body, and reaches thus the cerebellum of the same side. That the anterior one-half of the bundle of fibres of the lateral column (anterior mixed tract) passes upwards to the level of the medulla, appearing to terminate in its lateral part at the reticular formation, where some fibres seem to terminate in cells, but the major portion cross the raphe and ascend behind the pyramids to the striate body (nucleus lenticularis and nucleus caudatus). That in all probability these lateral columns contain secretory, vaso-motor, and other fibres, which are continued doubtless into the medulla. That the fundamental portion of the posterior columns of the cord (columns of Burdach) pass upwards, and largely bending beneath the floor of the fourth ventricle, ascend, and as they do so approach the raphe, decussate for some distance up the raphe, and then taking a position behind the representative in the medulla of the corresponding part of the anterior column of the cord, they pass to terminate chiefly in the thalamus opticus. Then again as regards the grey matter, it is continued upward from the cord into the medulla, much modified in form and situation, but still clearly recognizable. The anterior horns of grey matter in the medulla are almost or quite severed from the central grey matter, and in this it differs from the cord. The anterior horn of grey matter, as it passes up through the medulla, is pushed backwards with the fundamental portion of the anterior white column which belongs with it, and it is more divided into separate portions than in the cord.

The posterior horns of grey matter are also cut off in great measure from the central grey matter, and divided into separate nuclei more or less clearly, and the whole posterior cornu of the medulla is carried outward and forwards, so as to occupy relatively in the medulla a very different position from what it does in the cord. Then the central grey matter which forms a tube in the cord, is in the medulla laid open from behind down to the central canal, and then spread out laterally so as to expose the central grey matter in the floor of the fourth ventricle on the back of the medulla. This laying open the grey tube down to the central canal, in that part of its course which corresponds to the medulla, and the rolling outwards of the divided edges, it will be readily seen, would impose certain changes in form of the medulla as compared with the cord, and certain changes in relative position of parts, which are found by actual investigation to be much as might have been expected *a priori.* By such an arrangement as we have adverted to, it would be expected that the medulla, as compared with the cord, would be wide from side to side, and thin antero-posteriorly. This is seen at once to be the fact.

The medulla has various nerves enter and implant their fibres in its cell groups, but this also happens to the cord, but in a less orderly manner in the former than the latter. The nuclei of the motor and sensory nerves of the medulla, are more distinct than those of the cord. But they are destined to act with more independence and in more complex relations than those of the cord, and hence their comparative distinctness. Then, again, the medulla contains vaso-motor, cardiac, respiratory, secretory and other important centres, especially situated in or near the floor of the fourth ventricle. But the medulla above the cord, as it is in rank in all physiological aspects, is not in much peculiar even here as compared with the cord. The cord also contains vaso- motor, secretory, respiratory centres, &c., though subordinate to those in the medulla. Then again, the medulla as compared with the cord has some special formations, such as the olivary bodies, by which its size and complexity are increased.

But the anatomical and physiological relations of these bodies (olivary) are too imperfectly understood as yet, to enable us to say to what extent they impose differences on the medulla, as compared with the cord. The olivary bodies are certainly very special structures and this points to their having very special functions. But we may recur to these bodies briefly again. Then, the medulla as compared with the cord, is provided with a very great number of commissural fibres, which implies what is known to be a fact, that the actions excited from the medulla are bilateral as a rule. But this state of things is not peculiar to the medulla, though far less true of the brain and cord. Its relations with the cerebellum especially, through the cerebellar fibres of the pons, are numerous and complex, but the cord also has relations with the cerebellum, especially through the restiform bodies,or portions of the same.

The special nuclei of the medulla,which so much abound, and the central massive portions of the grey matter of the medulla have very extensive and complicated relations with the cell groups above in the basal system and cortex of the cerebrum, but there is no reason to suppose they differ fundamentally from those believed to exist in the cord.

(*To be continued.*)

III.—HISTOLOGY OF PERIPHERAL NERVES.

I. Leçons sur l'Histologie du Système Nerveux, par M. L. Ranvier. (*Lectures on the histology of the nervous system.*) Paris : F. Savy, 1878.

II. Axis Cylinder and Ganglionic Cell. By Hans Schultze. *Archiv f. Anatomie und Phys.*, 1878. Heft IV. and V., pp. 259.

III. Histology of Nerve Fibre and Axis Cylinder. By Th. Rumpf, in *Untersuchungen aus dem Phys. Institut der Universitaet Heidelberg.* (*Researches made in the Heidelberg Physiological Laboratory.* Edited by Dr. W. Kühne.) Heidelberg : Carl Winter, 1878.

I. The course of teaching pursued at the "College de France," at which L. Ranvier is Professor, differs from the plan of other institutes. It is intended not only to instruct students, but also to stimulate them to new researches. This merit is not the least important of Ranvier's work. This book brings the subject up to date in all its details, without, however, bearing the aspect of an encyclopedia. On the contrary, every new statement and every confirmation of the work of others bears the stamp of Ranvier's genius. The student, without previous knowledge of the subject, can not only follow profitably the work to the end, but he can also reproduce each result stated without previous preparation, such is the preciseness with which every method is detailed. The book besides abounds with suggestions; it is one of that rare kind which one cannot read without being stimulated to thinking. Necessarily there is a certain verbosity which may seem superfluous to the adept, but to which the work owes in part its easy reading and grace of style. The plates accompanying it render the book a work of art.

The introductory lecture treats of the evolution of the nervous system. In an amœba or a leucocyte we see an organism endowed with sensibility and motility. The globule, round and smooth when undisturbed, sends out prolongations and processes when irritated by contact of a foreign substance, but only on that side where it is touched, hence motility is evidently preceded by a sensation. The first *differentiation* into motor and sensory parts we find in the cells of the hydra as described by Kleinenberg. This animal has the shape of an open bag ; the wall surrounding the internal cavity consists of three layers of cells called in analogy with embryonic nomenclature, ectoderm, mesoderm, and endoderm. Each cell of the outer layer is really continuous with a cell in the middle layer, the two together constituting the neuro-muscular cell of Kleinenberg. In the outer half resides the sensibility of the animal, while the inner part of the cell serves the purpose of motor elements. Further differentiation of this apparatus gives gradually the schema of the nervous system of vertebrates.

Commencing hereupon the description of medullated nerve fibres, Ranvier combats the old view of the coagulation of myeline after death. From the end of a divided living nerve, examined in a drop of water, the myeline escapes slowly in curly streaks, and, after liberation, assumes the form of irregular globules without showing any signs of coagulation. This escape is not caused by the elasticity of the sheath of Schwann. This membrane folds itself over the axis cylinder, after the escape of the myeline, in such a manner as to show that it is neither very elastic nor stretched during the normal state. The escape is more likely due, according to Ranvier, to the hygroscopic properties of the myeline. The same phenomenon can be observed as well in a fluid less destructive to live cells than water, for instance, serum containing a trace of iodine. From the action of water the axis cylinder swells and becomes granulated.

Rumpf (III.) assigns a different cause for the escape of myeline. According to him the motive power is to be sought in the swelling of the axis cylinder by the action of water, which enlarging presses out the myeline. To prove this he dissolved the axis cylinders of fresh nerves by agents not altering the myeline, and on placing the specimen hereupon in water no escape of myeline occurred.—In the next place Ranvier studies the action of coloring agents. On placing a teased nerve into picrocarmine the coloration of the axis cylinder commences at the free end ; it proceeds also from the annular constrictions where the myeline is wanting. Where a fibre is accidentally bent in the form of a loop, the axis cylinder is pressed against the sheath of Schwann at the convexity of the bend displacing the myeline ; at such points also the coloration is more marked. From this can be deduced that the sheath of Schwann is permeable to solutions of crystallizable bodies—picrocarmine—while the myeline is not. On treating thin nerves while still in place with a solution of nitrate of silver— 1 to 3 parts in 1,000—small crosses appear here and there after reduction of the silver by light. These correspond to the annular constrictions described some years ago by Ranvier. The short horizontal arm of the cross is a black line, indicating a thin layer of cement, interposed between the ends of the sheath of Schwann of two contiguous interannular segments. Hence, the sheath of Schwann of a fibre consists of as many segments as there are interannular divisions. The long vertical arm of the cross is formed by the axis cylinder blackened by the silver to a short extent on each side of the constriction. The agent evidently attacks the axis cylinder at the constriction, where no myeline exists, and extends its action a short distance either way. Higher powers of the microscope show that the axis cylinder is not blackened *in toto*, but that black cross-lines alternate with clear spaces—the striæ described by Frommann. Of these the author gives no further explanation.

The striæ of Frommann appear in a different light from the researches of Rumpf (III). In the same volume (Researches from the Heidelberg Laboratory) there is also a short note by Morochowetz on the action of silver, which confirms and extends Rumpf's remarks.

According to these observations the silver lines do not belong to the axis cylinder itself, but to the sheath of the axis cylinder —the internal horn-sheath—(which will be noticed in detail in a later part of this review). This can be shown by dissolving the axis cylinder after reduction of the silver—by which treatment the striæ are not altered. Rumpf succeeded also in coloring with silver nerves from which, by a method to be described hereafter, the axis cylinder had been removed during life, and still the silver striæ were produced. The black lines form a series of rings around the axis cylinder (or its sheath), which exceed slightly in diameter the thickness of the axis cylinder and sheath, as is apparent even in the original plates of Frommann (*Virchow's Archiv*, vol. 31). They correspond evidently to circular spaces around the axis cylinder filled with substances having an affinity for silver.

At the point of the annular constriction Ranvier describes a very regular bi-conical thickening of the axis cylinder. In his preparations this adhered to the axis cylinder when the latter was torn from the sheath of Schwann. Morochowetz, however, saw the same adhering in some preparations to the sheath of Schwann ; in the latter case it had the appearance of a black circular disk forming a partition between the sheath of Schwann of two interannular segments, and perforated in the centre to give passage to the axis cylinder. Most probably this thickening or disk is identical with the layer of cement forming the short horizontal of the cross produced by nitrate of silver at the point of constriction.

Discussing the mode of employing osmic acid, Ranvier insists on keeping the nerve in its natural state of tension during the action of the agent. Nerves not stretched shrink and present altered appearances. He likewise insists that in the mounting in glycerine the latter fluid ought to be added very gradually if one wishes to avoid artificial deformations. Describing detailedly the annular constrictions, as seen after the action of osmic acid, he mentions that the sheath of Schwann is slightly dilated before its involvement in the constriction, hence a thickening of the coat of myeline at the ends of the segments. At the point of constriction the myeline is *always* interrupted. The apparently incomplete constrictions described by other authors are artefacts, as can be shown by compressing the nerve at some point while it is immersed in osmic acid. It is then found that the pressure displaces the semi-fluid myeline and causes it to rupture the attachment of the sheath of Schwann to the bi-conical thickening of the axis cylinder at the constriction of Ranvier. Hence the artificial appearance of continuity of the myeline between two segments. Under the influence of osmic acid the myeline coagulates and becomes brittle; the hardened myeline breaks very easily, and at the point of rupture the sheath of Schwann can be more distinctly traced by the aid of any other method.

Each interannular segment possesses one nucleus, situated on the inside of the sheath of Schwann at or near its centre. It is surrounded by a thin coating of protoplasm, more voluminous in the young than in the adult. Very distinct preparations are obtained by coloration with picrocarmine after a short sojourn in osmic acid.

The appearances of cross-sections of hardened nerves are next described. During the hardening process the nerve is maintained in its normal state of tension. The axis cylinder of nerves hardened in chromic acid has a star-shaped appearance. This is an artefact; under the influence of chromic acid the myeline hardens in the form of globules, the convexity of which leaves its imprint on the contours of the soft axis cylinders. Other hardening agents do not produce this distortion. In the cross-section the axis cylinder is seen to be surrounded by a thin sheath which is not colored by carmine. The same appearance is found when an axis cylinder is isolated by teasing in osmic acid. This corresponds to the axial sheath described by Mauthner, also Remak, Stilling and others. Klebs has asserted that the axis cylinder is surrounded by a periaxial free space. This, however, is an artefact; in reality there exists a sheath which shows no structure (except the rings developed by silver). In cross-sections differences are found between different fibres; this, however, depends on the place at which the nerve is cut. If the section passes through an annular constriction, the space between the axis cylinder and the sheath of Schwann is not filled with myeline, which, of course, *is* present when the section passes through any other point. But in many fibres seen in cross-section the myeline seems to be divided into two concentric rings by a clear line. This appearance is due to the segmental structure of the myeline as described by Schmidt (and independently of him by Lauterman and by Zawerthal). But this will be better understood after giving a *resumé* of Ranvier's view of the structure of medullated nerves.

Ranvier considers each inter-annular segment as the equivalent of a single cell, which he compares to an elongated adipose cell pierced in its centre by the axis cylinder. The sheath of Schwann he considers as the cell membrane, the contents of which are the nucleus, situated excentrically, and myeline. Between cell membrane and myeline he claims the existence of an extremely thin layer of protoplasm, as it is also found in the fat cell. This protoplasmic layer, lining the sheath of Schwann, passes over to the axis cylinder at the point of constriction, and thence returns to the other end of the segment as the sheath of the axis cylinder. From the latter protoplasmic partitions pass in an oblique direction to the protoplasm lining the sheath of Schwann, and the portions of myeline separated by these partitions constitute the segments of Schmidt. In the isolated nerve fibres these segments of Schmidt appear as cylinders of unequal length, one end being conical and fitting into the corresponding concave end of the adjoining cylinder. If a cross section be made through the junction of two myeline segments, the protoplasmic partition is seen as a clear ring, with a concentric ring of myeline on either side of it.—This view appears original and striking, but Ranvier himself does not consider it more than a hypothesis. It is true that the supposed protoplasmic nature of this lining layer has only been demonstrated around the nucleus, but the protoplasm is ordinarily also not visible in the circumference of adipose cells, unless swollen from œdema.

Much light has been thrown on the chemistry and structure of these lining layers by the researches of Kühne and Ewald. ("Die Verdauung als histologische Methode." *Verhandl. d. Naturh. med. Vereins zu Heidelberg.* Sep. Abdr. Nov., 1876.) Although they were published before R.'s book was completed, they are not quoted by him.

These authors dissolved the myeline of peripheral nerves and sections of the spinal cord in boiling alcohol and ether, and digested hereupon the remnant with a solution of *trypsin* (pancreatin). There remained after digestion two sheaths, one within the other, with a trellis-work of the same material uniting them. The inner sheath is evidently the tunic of the axis cylinder, while the outer sheath encases the myeline, separating it from the sheath of Schwann.

The material of which these "horn-sheaths" are composed is called by Kühne "neurokeratine." With the aid of the same method E. and K. also discovered that there exists on the outside of the sheath of Schwann a thin coating of longitudinal collagene fibres. The sheath of Schwann itself is dissolved by trypsin.

Further details resulting from these investigations have been furnished by Rumpf. Instead of ether and boiling alcohol, hot chloroform can equally well be used to dissolve the myeline in order to demonstrate the horn-sheaths and trabeculæ. Although visible after this procedure alone, they gain in distinctness by subsequent digestion of the axis cylinder by trypsin. Another method is furnished by dissolving myeline and axis cylinder in glycocholate of sodium (bile). Perhaps the least objectionable procedure consists in the expulsion of the myeline by the swelling of the axis cylinder from the action of distilled water. Prolonged maceration in water (for twenty-four hours) finally *dissolves* the axis cylinder entirely. The two horn-sheaths are then seen as concentric tubes, one within the other, the outer one being quite distinct from the sheath of Schwann. Thick trabeculæ pass at intervals from one to the other, in an oblique direction. Usually there are three trabeculæ radiating at once level from the inner sheath, thus forming a series of incomplete partitions which divide the myeline into the segments of Schmidt. The statements of Ranvier as to the existence of a lining of the sheath of Schwann continuous with the sheath of the axis cylinder, are hence supported by these preparations. Even his assertion that this system of sheaths is protoplasmic in nature is not disproven. The sheaths, as demonstrated by maceration in water, do not consist alone of neurokeratine, but contain besides albuminoids. In proof of this they can be colored by Millon's reagent. Furthermore, they become reduced in thickness by digestion with trypsin, which dissolves the albuminous portion. The inner horn-sheath covers the axis cylinder continuously, passing with it through the constrictions. The outer sheath is also uninterrupted in its course, but constricted together with the sheath of Schwann at the level of Ranvier's annular constrictions. The myeline is undoubtedly interrupted at this point, probably by the disc of cement which becomes blackened when treated with silver. This bar, however, of cement can easily be forced by violence.

Rumpf has made some important researches in the chemical nature of the axis cylinder. By an intense red coloration with Millon's reagent, he demonstrated positively the albuminoid character of this structure. The fresh axis cylinder is soluble in distilled water, bile, and still more so in a 0.1 per cent. solution of potassic hydrate. By the action of alcohol, however, it is coagulated, and in this state insoluble in the above reagents. Coagulation is also produced by a temperature of 50 to 52° C. (nerves of the frog).

In order to search for myosin—the albuminoid of muscles—in the axis cylinder, nerves were placed into solutions of NaCl. of the strength of 5 to 10 per cent., in which myosin is soluble. Such fluids, however, did not dissolve the axis cylinder, but on diminishing the concentration the astonishing observation was made, that water containing less than one per cent. of salt removed the axis cylinder completely. Hitherto a 0.75 per cent. solution of NaCl. had always been considered an indifferent fluid for nerves! Nevertheless, this dissolves the axis cylinder completely within 72 hours without producing any other changes in the remaining structures. Weaker solutions produce the same result still faster, but cause the axis cylinder to swell in the first place and hence force out the myeline. This unexpected observation induced Rumpf to test the solvent action of lymph, which contains the same percentage of salt as the hitherto supposed "indifferent" fluid. To his astonishment he found *the axis cylinder of excised frog's nerves completely dissolved in frog's lymph* within three days! Further experiments of Rumpf relating to the persistence of the axis cylinder during life will be related further on.

The fibrillary structure of the axis cylinder, as described by Max Schultze, is not mentioned by Ranvier in this place. Later on, however, he declared himself partisan of that theory, which, although he found no evidence of it in ordinary nerves, he could satisfactorily prove in some other cases. In the central end of divided nerves (Vol. II., p. 30) the axis cylinder swells—becomes hypertrophied as R. calls it—and in this state shows a distinct striation—an appearance which could only be caused by a bundle of fibrils. Another instance of distinct striation was found by Ranvier in the nerves of the electric organ of the torpedo (Vol. II., p. 125). In this organ the medullated nerves present a vague striation, their unmedullated twigs, however, are seen distinctly to consist of fibrils. In this instance the branches together are often more voluminous than the axis cylinder of the nerve fibre from which they arise. As a cause, Ranvier observed distinctly that the ultimate fibrils of which these branches consist pass from one branch into another without entering the larger fibre from which the branches arise. Thus there is formed a system of loops connecting the different nerve terminations.

Positive demonstration of the fibrillary structure of the axis cylinder was furnished by Hans Schultze (II.). Fresh nerves observed in iodized serum as well as in other fluids, show a decided striation of the axis cylinder. A particularly good object was found in the spinal roots of the frog. The most conclusive preparations were obtained by the use of very dilute solutions of osmic acid or nitrate of silver. In these the end of the axis cylinder protruding beyond its sheath seems to split up like a camel's hair pencil into separate fibrils. By careful adjustment of the focus the fibrillæ could be traced along the course of the axis cylinder. The plates accompanying Schultze's article are perfectly convincing—provided they are not exaggerated. Schultze claims that the fibrils are imbedded in a granular mass. The same appearances he could also trace in the transparent tail of the living embryonic salamander.

Ranvier has also examined living nerves in the lungs of the frog distended by the apparatus of Holmgren. Under these circumstances no fibrillation was seen by him. But he strongly insists on the fact that the living nerve thus displayed, shows the difference between the axis cylinder and myeline, as well as annular constrictions and the segments of Schmidt, in a perfectly unequivocal manner.

In the description of non-medullated fibres (Remak's fibres) Ranvier points out the confusion of authors in not distinguishing between connective tissue fibrillæ and Remak's nerve fibres. Both show a longitudinal striation; but the former have the appearance of a bundle of wavy hairs, running parallel, while the latter form a net-work with elongated meshes of anastomotic tendrils of unequal thickness. The thickness, however, depends on the number of ultimate fibrils of which the coarser fibres are composed. Each ultimate fibril has a nucleus on its surface with a thin protoplasmic coating. No sheath can be demonstrated around the fibres. Remak's fibres are colored orange by picrocarmine, which does not stain connective tissue fibres. But the most certain test for non-medullated nerve fibres is maceration in bichromate of ammonium, which produces such numerous vacuoles, as to give the fibres the appearance of pearl strings.

The description of the connective tissue of nerves is preceded, as indeed are all chapters, by a short but judicial historical review. Like his immortal master, Claude Bernard, Ranvier does not content himself with simply quoting and throwing overboard the results of authors whom he is forced to contradict, but seeks the cause of their errors in the conditions of their experiments. Isolated nerve fibres in their course through muscles are seen to be enclosed by a sheath, which Ranvier calls after its discoverer, the sheath of Henle. It seems to be structureless, but is lined on its internal surface by endothelial cells, not forming a continuous layer. Besides these cells two other kinds can be recognized, viz., independent connective tissue cells, situated far apart between the sheaths of Schwann and of Henle, and flat connective cells on the outside of the sheath of Henle. Small nerve-branches, containing several fibres, possess a similar sheath, in the inside of which, however, the endothelial cells form a continuous layer. As the trunks increase in size, their sheath is transformed into a structure, called by Ranvier "lamellar" sheath. In its description Ranvier becomes quite polemical. Robin is charged with an incorrect, in fact, imaginary description, while Axel Key and Retzius, who agree with our author, are accused of ignoring R.'s priority, since in 1872 he published his first article on the subject, an article which remains undisputed.

The "lamellar" sheath consists of a series of thin, concentric layers, varying in number with the thickness of the nerve. Each layer is formed of a trellis of connective fibres, with some elastic fibres woven in and lined on both surfaces with endothelial cells. Each layer is perforated by irregular windows. Trabeculæ pass from one layer to another, so as to give the whole considerable strength. From the centre (innermost layer) to the periphery the layers increase in thickness, until they finally blend with the areolar tissue surrounding the nerve trunk, the perifascicular connective tissue, as it is called by Ranvier. Under the name intrafascicular connective tissue, he designates the areolar frame of the trunk itself. The inner layers of the lamellar sheath send irregular partitions into the trunk; from these secondary branches proceed and subdivide further. The ultimate elements of which these are composed are connective tissue fibres and flat cells. Each nerve fibre is finally surrounded by a sheath of longitudinally arranged connective tissue fibrillæ. This description corresponds with the results obtained by Ewald and Kühne with the digestion method.

The vessels of the nerve trunks pass along in the sheath and connective tissue partitions to the inside of the trunk. Around the nerve fibres the capillaries form a characteristic net-work with elongated meshes. The capillaries have the shape of loops, from the convexity of which other capillaries arise. They are covered by an adventitial sheath consisting of flat cells. Lymph-vessels do not exist in nerve trunks; they are only found in the perifascicular tissue. In the trunk the lymph finds its way, as can be shown by injections, between the interspaces of the areolar framework and of the lamellar sheath.

To illustrate some points in the nutrition of nerves, an interesting experiment is next shown by Ranvier. In a living rabbit the sciatic nerve is exposed, and the wound filled with pure tepid water, which is repeatedly renewed. In the course of twenty minutes, that portion of the nerve which has been bathed in water has lost both excitability and conductility. Examined after treatment with osmic acid, it is found that the water has entered at the constrictions and crowded back the myeline ; the dilated pocket of the sheath of Schwann at the ends of the segment contains a fluid, in which fine granulations are suspended. The axis cylinder is swollen, as are also the partitions of Schmidt. If a 0.5 per cent. solution of NaCl. is substituted for the water the nerve does *not* lose its properties, even after 5 hours. Nevertheless, the microscope reveals the same changes, with the interesting addition of a distinct striation of the axis cylinder. The pure water must hence have produced invisible changes besides these, to account for the paralysis. The action of water, moreover, is permanent ; a nerve destroyed by it does not recover its properties, but degenerates as after section.

Ranvier's description of the degeneration of divided nerves is, no doubt, the most complete article on the subject. Bakowiecki has recently maintained, that previous attempts to prevent degeneration by a suture of the divided nerve failed on account of the material employed. In his own experiment he claims to have obtained anatomical as well as physiological union by the use of a catgut suture. Ranvier, however, denies the accuracy of these statements, on account of a total want of success in his own experiments.

The rapidity with which the peripheral segment of the divided nerve loses its properties depends on the energy of life. The time is slightly shorter in young, than in older animals. While in the cold-blooded frog the nerve retains its motility for thirty days, and in the sluggish plagiostoma even more than six weeks, the peripheral stump becomes unexcitable within four days in dogs, three in pigeons, and two in rabbits. Ill-health also retards the time. The animal which has suffered from the wound necessary for the section of one nerve, retains in this state the properties of a second nerve, now divided, a few hours longer than when normal. Some experiments, however, to retard the time by bleeding the animals, did not succeed.

In order to avoid any confusion between the histological changes of degeneration and the cadaveric alterations of nerves, Ranvier devotes a short paragraph to the details of the latter. Twenty-four hours after death the myeline has receded slightly from the ends of the interannular segments, and the sheath of Schwann here encloses a clear fluid. Beyond this, however, the nerve appears unchanged, and remains so for about 12 days. It is only after the lapse of this time that the myeline becomes opaque and charged with fatty granules.

The peripheral stump of a nerve examined within one hour after its division, shows the myeline escaping from the point of section. The myeline is swollen, opaque and granular in appearance. Engelmann recently claimed, that this process did not extend beyond the first constriction in either peripheral or central stump. Guiseppe Colasanti (*Arch., f. Anat.* Heft III. and IV., 1878, p. 206,) confirms this statement. According to his description the entire nerve—as far as the first constriction—consists of a strongly refractive, apparently homogeneous mass. He proposes to call this alteration, traumatic change of divided nerves.

Ranvier admits this limitation of the process in frogs, but claims that in mammals it does extend beyond the first constriction ; yet he does not describe any further details. Besides this alteration of the nerve fibres, red and white blood globules are found at the point of section. The leucocytes are infiltrated with myeline and fat. From analogous experiments on the omentum of Guinea-pigs, Ranvier admits that the flat cells of the intra-fascicular connective tissue can be transformed into leucocytes.

Twenty-four hours after the section changes begin in the peripheral portion of the nerve. The nuclei of the interannular segments become hypertrophied and their nucleoli more marked. The protoplasm surrounding the nucleus is also swollen and can be seen to line the sheath of Schwann in its entire extent.

After the lapse of fifty hours these changes have progressed (in the rabbit). The intense swelling of the protoplasm now displays the protoplasmic nature of the partitions of Schmidt. By their enlargement the myeline becomes divided into fragments. At other points, however, especially around the nucleus, the protoplasm forms projections, which cut through both myeline and axis cylinder, and finally, fill completely the sheath of Schwann. The myeline continues to divide still further, until the whole nerve tube becomes filled with minute drops, partly myeline, partly fat. The oleaginous portion of the myeline is changed partly into a soluble soap, which is absorbed by the protoplasm, and redeposited in its interior in the form of oil globules.

What becomes of the axis cylinder ? This question has been variously answered by different observers ; indeed the solution is very difficult. The coloration of the axis cylinder is not obtained easily on account of the impermeability of the myeline to coloring fluids. The best results were obtained by Ranvier by maceration in bichromate of ammonium. This hardening fluid produces fissures in the myeline, through which the coloring agent—picrocarmine—can reach the axis cylinder. The latter is now seen—divided into fragments of unequal length, by an occasional projection of protoplasm. The fragments are often spiral in shape, evidently from pressure of the segmenting myeline.

About the fifth day the nuclei begin to multiply. The nucleolus enlarges in the first place, gets the shape of a figure 8 and then divides. Hereupon the nucleus undergoes the same changes. The two nuclei thus created subdivide into four, and so on. This multiplication of nuclei ceases, however, within some days.

About the 12th day the nerve tube is filled with drops of myeline, nuclei and an intermediate substance. The myeline drops were not stained uniformly by osmic acid, some of them being scarcely colored. Since the dark coloration produced by osmic acid depends on its combination with fats, it is probable that these have been dissolved out of the colorless drops. The intermediate substance consists of protoplasm, which appears striated longitudinally.*

From this time onward, until about the seventy-second day, the nerve changes scarcely any in appearance.

The unmedullated nerve fibres also undergo certain changes after section of the nerve. Their nuclei enlarge and become filled with several nucleoli. Some nuclei have the shape of a figure 8. As to their multiplication, however, Ranvier is silent. In the interior of the fibres small vacuoles appear. These are replaced within a few days by fat-granules. Myeline drops are never seen.

As regards the connective tissue of nerves, no change is found in the fibres. The flat cells, however, enlarge, or at least become more distinctly visible, and become studded at first with vacuoles, and subsequently with fat granules. Myeline drops are found in their interior only at the point of the section. Later, about the twentieth day, the cells attain a globular shape and are infiltrated with some substance colored uniformly bluish-grey by osmic acid. The endothelial cells of the vessels undergo the same changes.

In short, we can say that the primary changes in the peripheral segment of a divided nerve consist in the growth of all the protoplasm contained, viz., protoplasm of interannular segment, of the connective tissue cells and of the vessels. At the same time it becomes infiltrated with fat granules, evidently derived from saponification of the fatty parts of the myeline. The destruction of the axis cylinder and myeline are only secondary. The loss of function coincides with the fragmentation of the axis cylinder by the encroaching protoplasm.

Meanwhile changes of a different nature have occurred in the

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* As regards the behavior of the "horn-sheaths" during the degeneration of peripheral nerves, no statements have as yet appeared. In grey degeneration of the cord they were found destroyed or at least rarified by Rumpf (Congress of South German Neurologists at Wildbad, May, 1878. *Centralblatt f. Nervenheilkunde*, 1878, No. 6, p. 141).

central end of the divided nerve. At the time of the section the myeline escaped from the nerve tubes, usually not further, however, than the first constriction; in fact even the interannular segment included in the section is not always completely emptied. The axis cylinders now hypertrophy and in this state show a distinct striation. The space between the axis cylinder and the sheath of Schwann is filled with drops of myeline and cells. These are partly red blood globules, probably pressed in; partly, however, wandering leucocytes. The latter are mostly surrounded by granulations of myeline and fat. Ranvier claims that they digest the myeline, in fact *eat their way*, and hence may pass upward within the sheath of Schwann, beyond the first constriction. In some instances they corrode even the axis cylinder. The changes are, therefore, not limited by the first constriction. Moreover, the axis cylinder appears hypertrophied, even beyond this point, while the nucleus and protoplasm of the next interannular segment are in a state of proliferation.

In the peripheral *end* on the other hand the axis cylinders have been destroyed, and at the end of a fortnight nothing is left but the emptied sheaths of Schwann.

The regeneration was studied by Ranvier on the pneumogastric nerve of rabbits, rats and Guinea-pigs. The animals were examined between the sixtieth and the one hundred and sixtieth day. At this time it can be observed that the central part of the nerve trunk is colored intensely black as usually by osmic acid. The central end, thickened, is lighter colored, as are also the peripheral end and the peripheral trunk of the nerve. The cicatricial portion becomes greyish from the action of osmic acid.

These different portions are next described in detail. The appearances, Ranvier divides into classes, viz.: ordinary and "bizarre," using the latter term because the phenomena are as yet beyond all explanation.

In the peripheral trunk examined about the sixtieth or seventieth day after the action of osmic acid, a large number of pale fibres, bearing nuclei, can be observed. These fibres are parallel and without anastomoses, hence, quite different from Remak's fibres. Other fibres have drops of myeline attached to them. There are, besides, regenerated fibres, with a thin coating of myeline, the entire fibre being very thin (four thousandth of a millimetre in diameter), and interannular segments very short, perhaps one hundred and fifty micromillimetres in length, while a normal segment measures about one thousand micromillimetres. A number of old sheaths of Schwann are filled with new axis cylinders, sometimes several in number, which are intertwined. Later, sometimes even at this time, new medullated fibres are found, not contained in old sheaths. They are of less than normal size, being ten micromillimetres thick, while their constrictions are three to four hundred micromillimetres apart. These tubes are often accompanied by a non-medullated or even very thin medullated fibre wound around them. Other fibres are seen without sheath of Schwann. Frequently nerve fibres are seen to divide at the level of a constriction. Between the different fibres oval masses are found, consisting of protoplasm and myeline and containing nuclei: they are nothing but portions of degenerated nerve tubes. One form of elements is still to be mentioned. These are fibres medullated in the greater part of their course, but entirely without myeline in some places.

The cicatricial segment is formed of a number of small nerve bundles, even though the trunk may consist of a single fascicule. These smaller fascicules are enveloped by delicate endothelial sheaths—in fact, enlarged sheaths of Henle. The bundles consist of both medullated and non-medullated fibres, the former augmenting with time. The fibres are mostly very thin, and by no means all parallel. To illustrate this irregular arrangement, Ranvier refers to an instance, where the central and peripheral parts of the nerve seemed to end in free buttons, without any cicatricial segment. There was, however, between them a delicate membrane, apparently of connective tissue. But on dropping osmic acid on this membrane, fine black streaks appeared uniting the divided nerve ends. On coloring this specimen and placing it under the microscope, with due care, it was found, that the apparent membrane consisted in reality of a net-work of nerve fibres, connecting the central with the peripheral end. Similar deceptive appearances were most probably the cause of the erroneous statements of Philipeaux and Vulpian, that nerves can be regenerated without union to a central nerve end.

In the central end of the divided nerve, no degeneration proper occurs at any time. It may be said, that the efforts at regeneration commence at the moment of the section. Examined after the sixtieth day, it is found that from the old hypertrophied axis cylinders, there proceed in a peripheral direction new nerve fibres—as prolongations, so to speak. Many of these fibres are non-medullated, others possess myeline, but usually only a thin coat of it, which is sometimes interrupted.

The fibres themselves are mostly very thin, but not all alike in thickness. Frequently there proceed two or more fibres as prolongations of an original axis cylinder. Many of these fibres subdivide again, but always at a point of constriction, if they possess myeline. Often one of the new fibres is wound around a thicker one. Corresponding appearances are observed in cross-sections. In these it can be plainly demonstrated that the fibres of new formation grow into the old persisting sheaths of Schwann. Sometimes as many as twenty-five new tubes can be counted in a single dilated sheath.

Under the heading "bizarre" appearances, Ranvier describes some very strange observations. Of the thin fibres of new formation, one is often wound around the other. Sometimes several are intertwined like a cord. Again a fibre changes its direction suddenly, and coiling up itself becomes recurrent. How far it pursues this recurrent course could not be ascertained. The strange appearances Ranvier refers to an excessive growth in an insufficient space. Another queer fact has been described by S. Mayer some years ago. Mayer claimed the occurrence of true nerve cells in the course of the regenerated fibres. Ranvier has seen similar appearances in the regenerated pneumogastric nerve. These objects appeared like bipolar cells interspersed in the course of non-medullated fibres. But, as they do not contain a nucleus, they cannot be considered as nerve cells.

A number of experiments are still mentioned by Ranvier, in which he divided a nerve at two points, leaving it, however, *in situ*. The intermediate portion was found to undergo the usual degenerative changes. These experiments were only made on warm-blooded animals.

Results of a different significance were obtained by Rumpf (III.) from double nerve section in the frog. As we have mentioned above, the axis cylinder of the excised frog's nerve is dissolved by lymph. Nevertheless a nerve-muscle preparation preserves its vitality for many hours, if kept in lymph or even in a 0.7 per cent. solution of NaCl. Hence Rumpf supposed that the connection of the nerve with the muscle preserved it from the solvent action of the lymph, in other words that the peripheral termination exerted a trophic influence as well as the central termination of the nerve. The loss of function does not occur in the frog's nerves until about the thirtieth day after their division, while lymph will dissolve the axis cylinder of the excised nerve in about seventy hours. But on dividing a frog's nerve at two points, so as to cut off the intermediate portion from both its central and peripheral termination, Rumpf found that the axis cylinders of the intermediate portion disappeared within three to four days!

The nervous distribution in the electric organ of the torpedo (T. Galvani) is next studied by Ranvier, since on account of its simplicity it can be considered a prototype of nerve terminations. The organ is described as semi-lunar in shape, consisting of a large number of pentagonal prisms, reaching from the skin of the back to the front of the animal. Each one of these prisms is divided into superimposed cases by horizontal partitions or lamellæ, the interspaces between them being filled with a gelatinous mass. After a careful historical review of the subject, R. describes his own methods of investigation. To avoid shrinkage he injects a one per cent. solution of osmic acid into the interior of the organ, thus maintaining the delicate lamellæ in their normal state of tension. The hardened object is hereupon placed into osmic acid until blackened. In rays and torpedos *the axis cylinders* as well as the myeline are blackened by osmic acid. Horizontal slices are hereupon removed with scissors and shaken in water until the lamellæ are isolated. The lamellæ are curved like the cornea, their dorsal side being convex, their ventral concave. The nerves enter on the ventral side. Examined in a drop of water with the ventral surface upwards, five different levels are easily recognized by adjusting the microscope, viz.: 1. a plane, in which the medullated fibres are seen; 2. a lower plane in which capillaries ramify; 3. the medullated fibres dip down underneath the vessels to form a third plane; 4. a plane apparently granular, corresponding to the nerve-terminations; and 5. a plane presenting round nuclei and granules. In the first three of these planes star-shaped cells with delicate processes are found. These cells belong to the mucous tissue contained in the space between the lamellæ and filling out any interspaces existing between the nerve fibres. Certain peculiarities are found in these nerves. They divide at first dichotomously, giving off both lateral and terminal branches. They are surrounded by a delicate endothelial sheath, which, however, R., for reasons to be detailed hereafter, does not consider as the analogue of the sheath of Henle of muscular nerves. This sheath divides with the fibres. Sometimes, however, two branches are surrounded for a short distance by one sheath. The constrictions differ from other nerves, inasmuch as they are apparently stretched, *i.e.*, the constricted unmedullated space between two segments is lengthened. The axis cylinder itself is blackened by osmic acid. The sheath of Schwann is applied very closely. As the branches divide they become successively thinner, and their interannular segments shorter. R. points out at this place, that according to a definite law, the distance between the constrictions increases with the thickness of the fibres. Finally the smallest branches lose their myeline. At a point of constriction the myeline ceases to surround the nerve any further, while the sheath of Schwann and the secondary sheath continue, but are applied closer. The sheath of Schwann probably proceeds to the end of the fibre, while the secondary sheath ends abruptly in a *terminal ring.* The naked axis cylinders display in this organ a distinct fibrillary structure, particularly after the action of osmic acid or staining with gold by Gerlach's method. The places of bifurcation are usually triangular. This is due to the fact that the branches collectively contain more ultimate fibrillæ, than the original axis cylinder. Evidently fibrillæ pass from one branch into another, connecting by loops the nerve terminations. The ultimate ramification of the naked fibres has been compared by Wagner to the shape of a stag's horn.

The parts so far described are really yet on the outside (ventral surface) of the electric lamella. They are surrounded by the mucous tissue. The greatest difficulties in observation commence only in the study of the electric lamella itself, and now stronger lenses are required. Since much diversity of opinion exists as to the structure of the lamella apart from the nerve terminations, R. elucidates this point in the first place. We notice here with pleasure an illustration of Ranvier's methodic preciseness and the fine results to be obtained therefrom. The lamella, isolated after an interstitial injection of osmic acid, is macerated in a mixture of one part of alcohol to two of water. Hereupon it is placed in a glass slide, ventral side upwards, and allowed to dry partially. Thus adherent it is immersed in alcohol and smartly brushed with a camel's-hair brush. The lamella is thus split unequally into layers, of which four can be distinguished: (1) a layer, called *nervous lamella*, consisting really of two parts, a superficial (ventral) layer containing the ultimate nerve terminations, and a deep layer composed of short *vertical* fibres (called by R. *electric cilia*) passing like a row of palisades towards the (2) *intermediate layer*. This also consists of two portions, a superficial part of a finely granular appearance, which is due to the electric cilia, and a deep layer, more coarsely granular, containing nuclei. These two layers correspond to the fourth and fifth planes recognizable by different *focal* adjustment in the surface view of the entire lamella. (3) A structureless, thin membrane, the dorsal lamella and (4) a connective layer consisting of a trelliswork of very fine connective fibres which support the entire lamella. Corresponding appearances are obtained by cross-sections.

For the study of the nerve termination three methods are recommended, the results of one supplementing and controlling the appearances furnished by the others. These are (*a*) staining with (solid) nitrate of silver, (*b*) intense coloration by osmic acid and subsequent treatment with chloride of gold and potassium (1 per cent.) and (*c*) staining with hematoxylin after preliminary injection of osmic acid. Satisfactory views are also obtained from the *fresh* lamella, but only after previous knowledge of its structure.

The different methods reveal, that the ultimate nerve fibres beyond the "stag's horn" ramification enter vertically the nervous lamella and expand here into *leaf-like terminations*, between which broad anastomoses exist. From all appearances R. concludes, that these terminations are held together by a structureless membrane, constituting the superficial layer of the nervous lamella. This membrane, which can even be isolated in small fragments, is in all probability derived from the union of the sheath of Schwann of the different fibres, since this sheath is not seen terminating at any other place. From the lower surface of the leaf-like nerve expansions the electric cilia pass like a regular row of palisades to the upper (ventral) surface of the intermediate layer. The lower portion of this layer is evidently not solid, since in the fresh specimen the coarser granules are seen to perform Brownian movements. The nuclei found in this layer possess no cell-body. R. hence considers the entire layer as the equivalent of a single cell with multiple nuclei.

The wall of the prisms of the electric organ consists of a trelliswork of connective tissue fibres. There is besides a framework running through the organ, in which the prisms are imbedded. This framework can be compared as regards its structure to the lamellar sheaths of nerve trunks. The electric lamellæ themselves are united with the wall of the prism by means of the network of connective tissue on their dorsal surface. The dorsal surfaces of all lamellæ (of one prism) are thus mutually connected. The remaining layers of the lamella bend downward along the wall of the prism and terminate just above the dorsal surface of the next lamella.

Next in place is a study of the electric nerve fibres as they appear in the trunk. Besides the sheath of Schwann the individual fibres are surrounded by a secondary sheath, which prolongs itself, and has been described as such with the fibres ramifying in the electric organ. Its existence in the trunk establishes a difference between it and the sheath of Henle of muscular nerves, which latter is really the continuation of the lamellar sheath. The interannular segments of the electric nerves are remarkably short. Thus a fibre in the electric nerve trunk of 12 micromillimetres diameter consists of segments of 500 to 600 micromillimetres in length, while fibres from other nerves of the same animal and of the same thickness consist of segments of twice that length. This observation supports R.'s theory, that the length of space between the constrictions varies inversely with the energy of nutrition of the nerve, since, according to R., the nutritive fluids pass in and out at the constrictions where no myeline opposes their passage. At any rate R. cannot well be contradicted in asserting that the electric nerves are the most active of any nerves in the sluggish torpedo.

The trunk of the electric nerve consists, near its origin, of a few large fascicules, which increase in number and diminish in size in passing towards the periphery. R., however, has never observed any division of nerve fibres within the trunk. The nerve bundles are surrounded by sheaths passing off from the common lamellar sheath. In the electric trunk all the intrafascicular tissue presents a more distinctly lamellated arrangement than it does in nerves of other animals. In other nerves the last trace of the intrafascicular tissue appears as a sheath around the nerve fibres, consisting of sparse connective fibres. In the electric nerves, on the other hand, we find instead, a sheath around the fibres in the trunk, consisting of lamellated endothelium, *i. e.*, the secondary sheath, which continues beyond the point where the ramifying nerves lose their myeline.

A peculiar mode of nerve division is observable in the walls of the electric prism. A single nerve fibre running on the inside of the wall will subdivide into a large number (as many as 20) branches. The axis cylinder undergoes a considerable thickening at the point of a constriction and splits abruptly into many branches. In honor of the discoverer, R. calls these divisions "bouquets" of Wagner. The resulting branches exceed in thickness the original axis cylinder, probably on account of the existence of loops of fibrillæ, which do not enter the parental axis cylinder. Still this is not so demonstrable, as it is in the non-medullated branches in the electric lamella, yet it can be positively observed that the volume of the collective nerve fibres increases from the place where they leave the trunk up to the periphery.

Ranvier enters hereupon into the physiology of the electric organ and furnishes us with a captivating hypothesis of its action. The organ is controlled by the will of the animal. In answer to a strong irritation the animal gives a severe shock. This shock, however, is composed, as Marey has shown, of a series of discharges, hence analogous to the muscular tetanus, consisting of superimposed single contractions. The shocks can be repeated, but feebler, until the animal is exhausted. Not every irritation causes a shock, unless the animal is enfeebled, when they become apparently involuntary reflexes. On laying bare the electric lobes, whence the electric nerves originate, discharges can be induced by direct irritation of the lobes. But a different result is observed when the electric nerves are divided and then irritated. *No shocks can be perceived by the hand*, although a delicate galvanometer or a sensitive nerve-muscle preparation will reveal a *very feeble* discharge following each direct irritation of the exposed and severed nerves. Boll recently stated, that the torpedo is not paralyzed by curare. R., however, found that strong doses of this poison *will* paralyze the motor nerves, without, however, impairing the activity of the electric nerves of the fish.

As a basis for his hypothesis, Ranvier reminds his readers that the electric lobes consist of nerve cells with two kinds of processes, (*a*) several short ramifying processes, (*b*) a single Deiters' process, which is prolonged as an axis cylinder. This axis cylinder continues in the trunk, until it splits in the wall of the electric prism into the bouquet of Wagner. From this point further subdivisions occur until the fibres end in the leaf-like expansion in the electric lamella. From these leaves the electric cilia proceed up to the surface of the intermediate layer. Let us suppose, that by some chemical process there occurs in the nerve cell a separation of electricity into positive and negative fluid, and that the positive electricity passes out into the branching processes. The negative electricity could in that case proceed along the Deiters' process and axis cylinder to the nerve termination and electric cilia. If now the intermediate layer is a poorer conductor than the other layers of the electric lamella, the dorsal surface will become charged with positive electricity. The dorsal surfaces of all lamellæ are in mutual connection through the walls of the prisms. The nerve terminations are also mutually connected through the nervous loops previously referred to. Hence the electric organ can be compared to a condenser or Leyden jar, which discharges itself as soon as the necessary electric tension has been attained. That the chemical processes generating the electricity do not occur in the electric organ itself, seems likely on considering the feeble blood-supply of the organ. On the other hand, their occurrence in the electric lobe is vouched for by the inability to obtain strong discharges by stimulating the divided electric nerves. The very feeble shock which does result from the direct stimulation of the nerve, might well be caused by chemical processes in the nerve itself.—This hypothesis rests, as far as histology is concerned, on a solid basis. It certainly deserves as much credit as any explanation of this organ by other authors.

The study of nerve terminations in muscles is preceded by a thorough resumé of the structure of the muscle. It would, however, unduly lengthen this review to follow Ranvier in this matter, as well as in his historical review of the nerve terminations. Suffice it to say, that these rather obscure subjects are treated in a remarkably clear manner.

Before proceeding to the demonstration of the ultimate nerve ends, Ranvier describes the mode of division of nerves in muscles, choosing as a type the thoracic cutaneous muscle of the frog. The animal is anæsthetized and extended upon a cork plate. Hereupon half a cubic centimetre of osmic acid (1 per cent. sol.) is injected under the skin over the sternum. The thin, transparent muscle, which passes from the latter bone upwards and outwards to the skin, is hereby rendered rigid and can now be easily removed, while in the fresh state this is a very delicate procedure. In the muscle examined in its nutrient fluid, the medullated nerves are seen to enter, several in number, at the lower corner of the internal surface. The subdivisions, very numerous, always occur at a point of constriction. The minute nerve trunk is enclosed by a thin lamellar sheath, which continues finally to surround the isolated fibres as a single layer of endothelial cells —the sheath of Henle. The sheath of Schwann is so closely applied to the nerve fibre that it cannot be recognized. The fibres terminate apparently with free ends. That this is a deception can be proven by the study of other muscles.

Commencing with insects, R. opens the shell of the leg of the hydrophilus and extracts some of the muscular fibres, which are then examined in a drop of the animal's abdominal fluid. As soon as the spontaneous movements cease, the double striation of the muscular fibres can be recognized. They are surrounded by tracheæ, which appear dark on account of the inclosed air, and by nerve fibres. The latter consist of an axis cylinder, presenting a fibrillary appearance, and are surrounded by a sheath peculiar to insects. At a certain place this sheath blends with the sarcolemma, while the axis cylinder enters the eminence of Doyère. The axis cylinder splits into a bundle of fibrillæ, which surround a granular cone, continuous with a layer of protoplasm lining the inside of the sarcolemma. The fibres cannot be followed beyond the base of the cone, where a number of nuclei can be seen.

Repeating as much as possible, in his demonstrations, the historical development of the subject, Ranvier chooses as the next specimen the muscles of the thigh of the lizard, treated with HCl. one part in 1,000 water. The agent coagulates the muscle and hence renders it opaque, but subsequently transforms the myosine into syntonine, and thus produces perfect transparency. In these preparations it can be seen that the sheath of Henle blends with the sarcolemma. It is not possible to ascertain what becomes of the sheath of Schwann. The axis cylinder cannot be followed beyond its entrance into the muscle, where it loses its myeline. Fifteen to twenty nuclei are found in the eminence, shrunken from the action of the acid.

Cohnheim's treatment with nitrate of silver is next resorted to. A fragment is taken with sharp scissors from the triceps muscle of the lizard and teased in a drop of the animal's serum. Those muscular fibres of the specimen which possess nerve terminations, visible with a low power, are transferred into a 0.2 per cent. solution of nitrate of silver. After ten to twenty seconds sojourn they are washed in distilled water and exposed to daylight. When browned by light they are transferred into dilute acetic acid (one per cent.) in which they regain their normal dimension after shrinking in the silver solution. They are now examined in a mixture of glycerine and water. By this procedure the living muscular fibres are stained brown, but on the surface only, while the portions underneath the nerves present a white negative image. It is thus seen that the nerve fibre divides, and that its branches subdivide still further, in a manner comparable to the branches of a tree (*arborisation*).

Supplementary positive images, in which the nerves are colored, while the muscle is pale, are obtained by staining with gold. Ranvier recommends for this Löwit's method, consisting in the successive treatment by formic acid (one in three), chloride of gold (one in one hundred), and again formic acid. Like all gold staining, the method is not always a success. The branching violet nerve fibres are seen to end in broad expansions. By the action of the formic acid, brances are often torn between a larger stem and the terminal expansion. The gold staining reveals sometimes a granular appearance surrounding the nerve branches.

Kühne, in a recent article (in *Untersuchungen aus dem phys. Institut Heidelberg*), claims, that during life anastomoses exist between the terminal expansions, which are but poorly shown by Löwit's method, on account of the rents produced by formic acid.

The intravaginal net-work of the muscle shown by gold staining, which Gerlach claims to be connected with the nerve, in fact, its termination, is altogether contested by Ranvier. According to him it is only a protoplasmic net-work studded with fat granules, and has nothing to do whatever with the nerve.

Further details are revealed by interstitial injections of osmic acid, followed by staining with picrocarmine. Cross-sections of muscle and eminence are also described by Ranvier. The results thus obtained are compared with the fresh specimen. Ranvier also mentions a procedure by which he succeeded in preserving the appearances of the fresh specimen. It consists in adding one part of alcohol in two parts of water on the glass slide.

From these different methods, applied to the muscles of the lizard and snake, the following results are obtained. The axis cylinder of the medullated nerve enters the muscle at the nerve eminence. At this point the myeline ends in a constriction, while the sheath of Henle blends with the sarcolemma. Sometimes the nerve branches immediately before it enters the muscle. The naked axis cylinder branches after entering the eminence, and ends finally with broad expansions. The anastomoses between the branches, which Kühne describes, Ranvier claims not to have seen. He refers them rather to the overlapping of branches. A granular *sole* at the base of the eminence does not exist, in reality the individual branches are surrounded by a hazy substance. Three kinds of nuclei are found in the eminence, (*a*) small, flat nuclei, strongly colored by picrocarmine, lining the membrane covering the eminence, hence really belonging to the sheath of Henle, and called by Ranvier *vaginal nuclei ;* (*b*) large, oval, double-contoured nuclei, with brilliant nucleoli—they are faintly colored by picrocarmine and are situated at the base of the eminence (*fundamental nuclei ;*) and (*c*) nuclei intermediate between these two varieties in size and coloration, situated between the nerve branches — *nuclei of nerve branches* (noyaux de l'arborisation).

Ranvier refers to some microscopic observations of curarized muscles, which did not differ from normal objects. Kühne, however, in his last memoir (*Untersuchungen, etc.*, Heft II., Bd. II., p. 208) claims to be able to distinguish between normal nerve terminations and those of lizards poisoned with *large* doses of curare. In the latter case the details of the branches appear more marked within the first hour after death than they do normally. During life no details can be recognized at all, all parts appearing homogeneous. It is only after excision of the muscular fibre that the details begin to appear gradually. According to Kühne the distinctness of the nerve eminence is so far in advance, in strongly curarized muscles, that a difference can be recognized.

In mammals the nerve terminations resemble those of the lizard, but are crowded into a somewhat smaller space.

In the frog the arrangement is somewhat different. The form of the branches resembles a bush, to which it has been compared by Kühne. The nerve fibres divide into several medullated branches applied to the sarcolemma like the extended fingers of a hand to the surface of a cylinder. Thence naked fibres pass underneath the sarcolemma and continue to branch, maintaining on the whole the form of a bush, with rather long branches, the ends of which are not broadened. Neither a granular substance nor fundamental nuclei are found, while the other varities of nuclei are similarly arranged as in lizards.

No method has as yet revealed any nerve prolongation beyond the apparent ends of the branches, hence we are justified in considering them the real nerve terminations.

The last pages of the book detail three observations made after the section of the sciatic nerve of rabbits. It was found that the degenerative changes, especially the fragmentation of the myeline, occurred earlier in the nerves near their termination than in the trunk. Ranvier seeks the cause in the shortness (and thinness) of the interannular segments, at the periphery of the nerve; since the changes depend on the activity of the cellular elements, they must progress the more rapidly the more there are of them in a given length. Probably there exist also differences in the nutritive supply of the nerve in the trunk and in the periphery. Twenty-four hours after the nerve section the nuclei of the nerve eminence were found enlarged, proving that they participate in the exaggerated activity of the protoplasm and nuclei of the nerve itself.

The object of this review was to abstract mainly those original observations, which have not yet passed into current literature. But apart from the wealth of new discoveries, the book is a mine of information both as to modes of research, and the results obtained. The style could not be excelled in clearness, and the plates, corresponding faithfully to the original slides, as the writer can testify, deserve the same praise. Perhaps the gravest fault to be found in the work is the scarcity of microscopic measurements. H. GRADLE.

IV.—LOCALIZATION IN BRAIN DISEASE.

LECTURES ON LOCALIZATION OF DISEASES OF THE BRAIN. By J. M. Charcot. Edited by Bourneville. Translated by Edward P. Fowler, M. D., New York. William Wood & Co., New York, 1878. Pp. 133.

This attractive little volume, composed of twelve lectures, introductory to his course in the faculty of medicine in Paris, are by the distinguished chief of the division for nervous affections, at the Salpétrière—Professor Charcot. They have been already published, in part at least, in the *Progrès Médical*, a Parisian journal edited by Dr. Bourneville (a pupil of Charcot), who charged himself with the duty of reporting and publishing them in France. These lectures were delivered at the opening of the course of instruction of the past year, and now appear for the first time in the English language.

Dr. Charcot is well known everywhere, among those interested in the study of nervous diseases, or who have at heart the true interests of scientific medicine. He is a good example, among living physicians, of a well balanced investigator, fairly comprehensive in knowledge, practical in aim, sound in judgment, simple in method, and capable not only of just and penetrating observation, but of safe generalization. Even his recent serious investigations into so unsubstantial a subject as *metallo-therapy* at the Salpétrière, singular as they may seem to be at a distance, do not require us to modify our judgment. Dr. Charcot is possessed evidently of a sound and versatile, though not a brilliant mind, one which does not incline him greatly to the paths of speculation, but rather to facts, and to inductions which do not wander far from their bases. Hence his writings, while clear, and their style free and direct, are not discursive nor voluminous.

Dr. Charcot draws in these studies, as in others, his materials from every available source — normal anatomy, experimental physiology, and clinical examinations, in his field of labor, giving special preference, very naturally and for good reasons as they seem to us to be, to clinical and pathological materials.

The attempt is made in these lectures to apply the doctrines of Hitzig, Ferrier and others to brain disease. His belief like theirs, is, that "the encephalon does not represent an homogeneous organ, a unit, but rather an association, or a confederation, composed of a certain number of diverse organs. To each of these organs belong distinct physiological properties, functions and faculties. Now, the physiological properties of each one of these parts being known, it becomes possible to deduce therefrom the conditions of a pathological state ; this being of course but a greater or less modification of the normal state, and not a result of the intervention of new laws." (P. 3.)

The first lecture is chiefly devoted to certain plain, and for his purposes clear statements as to the general arrangements of parts in the internal structure, and external configuration of the brain. These and other lectures are elucidated by schematic drawings, which are a great help to the reader. The next two lectures are almost wholly given to a description of the structural and topographical anatomy of the cortex of the brain.

In the fourth lecture a parallel is drawn between spinal and cerebral lesions, with the very judicious purpose of applying our present knowledge of the localization of spinal disease, to a study of localized brain disease. The latter part of this lecture, and the fifth, sixth, seventh, eighth and ninth lectures are given to a study of the peculiarities of distribution of the blood-vessels to different parts of the brain, and to the effects of disease of the same on the brain structure proper. In this part of his course he follows after Duret and Heubner, in their independent and admirable researches into the vascular supply to the encephalon. This is one of the most suggestive parts of the book.

The tenth lecture, is devoted to : cerebral hemi-anæsthesia (continued from the ninth lecture,) crossed amblyopia, and lateral hemiopia.

The eleventh lecture is given to a very suggestive discussion as to the "origin of the cerebral portion of the optic nerves."

Finally the last lecture (twelfth) is devoted to "secondary degeneration," and its bearing on the question of localization. Due credit is given to Tuerck, of Vienna, through whose labors these "degenerations" were first brought to light.

Such is an outline of this remarkably instructive little book. We are only prevented from entering on an extended and critical analysis of its contents, by the fact, that nearly every phase of the subject has been discussed in one way or another, in the pages of our journal, since 1874, so that our readers are already so far acquainted with the matter of the lectures as to make an extended notice superfluous. But to all who desire a clear and easily comprehensible outline of the present state of cerebral pathology, especially as it bears on localization of function in the brain, and from the hand of a master—we can most heartily commend these lectures.

We bespeak for them a wide circulation among physicians on this side of the Atlantic. We would not close without saying that the somewhat thankless labor of the translator appears to be well done.

V.—FOTHERGILL: ANTAGONISM OF MEDICINES.

The Antagonism of Therapeutic Agents: and What it Teaches. The Essay to which was awarded the Fothergillian Gold Medal of the Medical Society of London for 1878. By J. Milner Fothergill, M. D., Edin. Philadelphia: Henry C. Lea, 1878. Chicago: Jansen, McClurg & Co.

A rational scientific therapeutics that shall redeem practice from the often reiterated and never satisfactorily answered charge of empiricism is one of the greatest desiderata of medicine. It is to meet this want that so many hasty generalizations and half truths have been enounced to form the basis of systems of more or less thoroughly developed quackery. Men are taken with even a pretense of scientific system, and so much has this been the case that an educated empiricism in the best sense of the word has almost been the sole refuge of those who were capable of estimating the real merits of the various so-called schools of medical practice. But empiricism has not been absolutely the sole resource of true physicians; thanks to honest research in physiology and pathology, some general, if not universal, principles have been reached, and therapeutics is becoming less purely empirical every day. The little volume before us is an evidence of this fact: it is a treatise on scientific physiological therapeutics, such as might be expected from the well known reputation of its author.

Dr. Fothergill needs no introduction to our readers; there are few English medical men whose names are better known in this country. In this, his latest work, he has taken up the subject of the physiological antagonism of toxic agents in their therapeutic application more especially, and has produced a book that can be read with pleasure and profit by every one who has to do with the treatment of disease.

He divides his work into seven chapters. The first is devoted to an account of the experimental investigations into the antagonisms of remedies by Frazer, Bennett, Harley, Crichton Browne, the author himself, and others. This is a necessary introduction to his memoir, but as the results of the experiments are accessible elsewhere in text-books or have been mostly given from time to time in other issues of this journal, it is not necessary to repeat them here. The practical bearings of these results are discussed in the second chapter, which deserves more detailed attention.

In this chapter the physiological indications and contra-indications of chloral, morphia, belladonna and strychnia are discussed in a very instructive way, and it will prove profitable reading to any one who wishes to use these agents intelligently. We have not seen the dangers in the use of chloral in certain cases where the respiration is involved, stated any where else as clearly as they are here, and the same may be said of the remarks on the use of strychnia in the same conditions. The remark quoted from Crichton Browne that the danger in the use of chloral hydrate is proportional to the development of the brain, is suggestive, but it's practical importance may perhaps be over-estimated.

The third chapter is a digression on the automatic motor nervous centres or, as our author calls them, the rhythmically discharging centres for the respiration and circulation, and is introductory to the two succeeding chapters, in which the actions of drugs upon these functions are taken up. In the first of these the action of digitalis on the circulation is the principal subject considered. It consists mainly of a reiteration of the author's views, as given in an essay published some years ago, and now out of print, with perhaps some additions of the more recent results of observations on the subject. This portion of the book was not included in the prize essay, but was added on its publication. We can refer the reader to no other equally concise and full statement of the indications of this drug.

The other medicines that act on the circulation that are mentioned here at any length, are belladonna and aconite, and the notice that is given to them is comparatively brief. Dr. Fothergill repeats the caution of Farquharson as to the use of aconite in the aged, and extols its utility in children.

The fifth chapter contains an excellent *resumé* of the physiology of respiration, with a review of the physiological action of the principal drugs affecting its nervous mechanism, and then a practical discussion of their remedial effects in conditions of disease. The chapter closes with a study of the effects of temperature in disorders of the respiration.

In the sixth chapter the practical value of the physiological antagonisms of medicines in cases of poisoning is taken up. Here the author gives an account of a number of recent very remarkable instances of the use of this antagonism, one of which is especially noteworthy, occurring in Dr. Fothergill's own practice, in which there was injected hypodermically a full grain of sulphate of atropia, with the result of saving the life of a woman already nearly moribund from a large dose of laudanum, the physiological effects on the circulation and respiration being all the time carefully noted. Almost equally remarkable cases collected from various sources of the successful use of the antagonisms of strychnia and calabar bean, and strychnia and chloral and other poisons, are also related.

The last chapter treats of the use of these antagonisms in ordinary practice, and contains a number of illustrative instances, well but briefly stated, but we cannot give an abstract of it. Indeed it is difficult to state in brief anything of the whole work; it is not such a one as can easily be condensed. We can heartily recommend it to our readers, and perhaps that is all that such a notice as this should be expected to do. A knowledge of its contents cannot fail to be a most valuable addition to the professional information of the average practitioner of medicine.

VI.—LIONET: GENERAL PARALYSIS.

DES VARIÉTÉS DE LA PARALYSIE GENERALE DANS LEURS RAPPORTS AVEC LA PATHOGENIE. Par le Dr. Camille Lionet. Paris, 1878. (*The Varieties of General Paralysis, in their Relations to Pathogenesis.*)

It is not very long since, that a rather remarkable editorial appeared in a leading eastern medical periodical, commencing as follows: "A strange and formidable disease is stealing upon this country, a disease whose march is slow, but sure, whose grasp is fatal to mind and body, whose touch defiles as well as destroys. This terrible disease is general paresis or paralysis. Thirty years ago it was hardly or at all known in this country; even yet a local case of it has probably not yet occurred in Louisville, in St. Louis, or in New Orleans. On the other hand, in Boston, New York, and Philadelphia it is far from uncommon. In England it has long been known, while in Germany and France it is frequent."

We need not ask a better excuse for a review of one of the most recent memoirs on this disease than the above paragraph. If, as is asserted, general paresis is slowly but surely stealing over this country, having already blasted the eastern and middle section with its defiling and destroying touch, but has, as yet, mercifully spared the Mississippi valley, it is a fact that should be generally known. If, on the other hand, the extract only illustrates a too common ignorance as to the conditions and prevalence of this most formidable of cerebral diseases, then this ignorance certainly needs to be enlightened.

There is, in our minds, no question but that general paralysis is often misunderstood, and the editorial quoted, though itself exemplifying this ignorance, is therefore timely, and inasmuch as its intention is good and its results cannot be bad, we ought not to make an example of it. When western, and for that matter eastern asylums also, show such various percentages of this disease in their classification of their inmates, it seems even that those whom we should think ought of all men to recognize the disorder, have very different ideas of diagnosis. Such a work as the present one is therefore a fit subject for notice in our columns, and we shall endeavor to give briefly its principal features.

The title of the work, "Varieties of General Paralysis in their Relations to Pathogeny," is itself suggestive of something comparatively new in the study of this disease. In all the classifications of insanity there is no species better or even so well established, both on clinical and pathological grounds, as a morbid entity, as is general paralysis. But the varieties of its mode of invasion, in its apparent origin, and in the character and severity of many of the symptoms, are such, that it seems as if it might almost constitute a morbid genus, containing several allied but distinct species. In view of these facts, Dr. Lionet was led to study the affection and its literature to ascertain whether there might not be recognized distinct varieties of the affection, depending upon its etiology. He therefore analyzed the literature of the disease and his own observations, and as a result he classes all general paralytics into two great classes: those self-originating in the individual, and second, those in the etiology of which heredity plays a great part. The first of these are the typical general paralytics; the disease, starting generally from some well marked irregularity of life and excesses of all sorts, runs a regularly progressive course, without remissions, and the patient dies in paralytic dementia within two, three, or four years. The second great class of general paralytics, admitted by the author, form the subject of the larger part of the monograph. After some remarks on heredity in general, he passes in review the opinions that have been expressed before him, on its influence in the production of this affection. Bayle, Calmeil, Pinel, Quatrefages (of Montpellier), Brierre de Boismont and Dagonet, are all quoted as supporting the view that heredity in general plays an important part in the etiology of general paralysis. But besides these, Aubanel, Baillarger, Lunier, and Legrand du Saulle, have insisted on the influence of hereditary congestive tendencies as predisposing causes over others, and especially over hereditary insanity, which, indeed, has by some been altogether denied to be ever a cause of this disease. Our author admits two varieties of general paralysis due, at least in part, to heredity. The first of these, general paralysis of congestive origin, usually occurs in individuals of so-called sanguine temperament, plethoric, and of apoplectic tendencies. The disease is characterized by its sudden onset, being preceded by a congestive cerebral attack, or, it may be, by only a period of congestion, without the *ictus*, and following this a longer or shorter interval, the cerebral disorder makes its appearance. Throughout the course of the disease the congestive attacks are apt to appear, either as a passive form with somnolence and even coma, or as apoplectiform crises. Remissions may take place, and, thanks to them, the fatal result may be deferred for five, six, or even eight years from the onset. These remissions, however, are more apparent than real, the patients never recover lost ground; the disease is only temporarily arrested.

It must be allowed that other causes than an inheritance of the congestive tendency may give rise occasionally to this variety; injuries, insolation, suppression of habitual or normal hemorrhagic fluxes, &c., may also serve as its points of origin.

The third variety, or the second in which heredity plays a part, is general paralysis of vesanic origin, and its characteristics are a long prodromic period and true remissions. In these patients the first symptoms are psychic, a more or less notable mental disturbance. There is generally, previous to the pronounced mental aberration, a prodromic period, which has been described by M. Jules Falret as the stage of exaltation. In it, the intellect is sometimes even more active and capable than normally, the memory is quickened, and the sensibilities are exaggerated. But at the same time, the subjects are unreliable, abnormal in their emotions, fickle and often absurd in their judgments, and liable to sudden, transient and causeless fits of violence. As this stage passes on to that of pronounced delirium, the actions and judgment become still more bizarre, they abandon themselves to their impulses, the delusions of grandeur appear, at first moderate and possible, and become more and more extravagant as the disorder progresses ; the physical and moral symptoms make their appearance, and the patient passes on to the ordinary advanced stages of the affection. These cases also have their remissions, and they are much more complete than in the other variety. Nevertheless they do not absolutely recover in these, there still remains some mental weakness, and though the duration of the disease is greatly prolonged to perhaps ten, eleven, or fifteen years, the fatal result is only delayed.

These, as we have very briefly described them, are the three varieties of general paralysis which the author seeks to establish. After their description and several pages on their medico-legal aspects, the remainder of the volume is given to the history of cases illustrating each variety, selected from the literature of the disease. The etiological conditions of these varieties may not be universally admitted, nor perhaps are they always constant and distinct, but they will be recognized, at least in part, by those who have had much experience with this class of patients. In some of Dr. Lionet's collected cases of the second and third varieties, the history of heredity is wanting; this, as he states, is difficult to obtain, but after all it may be too much of an assumption to consider the hereditary tendency always as existing.

In the third variety there are some instances given in which an almost complete recovery took place, and no history of a relapse. These are, however, exceptional, and do not affect the rule as to the unfavorable prognosis. We may say also that, according to the statements of L. Meyer, there is some hope from energetic revulsive treatment in many cases of general paralysis, he having reported seven or eight cures out of fifteen cases treated. This our author does not mention. At the close of the volume the author gives his general conclusions, which we reproduce as summing up the memoir in brief:

1. General paralysis represents a pathological species.

2. Most frequently it arises of itself, without hereditary predisposition.

3. When hereditary predisposition exists the disease presents itself under one of two forms: congestive and vesanic.

4. The hereditary general paralyses of the congestive type are cases of brain disease before they become cases of insanity.

5. The hereditary cases of the vesanic type, are insane from the beginning.

6. There are therefore three varieties of general paralysis:

(1.) General paralysis of individual origin.
(2.) General paralysis of congestive origin.
(3.) General paralysis of vesanic origin.

7. General paralytics of individual origin never have any remission; and the duration of the disease is only 2 to 4 years.

8. General paralytics of congestive origin are especially remarkable from the frequency of their congestive attacks. They have remissions which are only temporary arrests of the disease. They may live 6, 7, or 8 years.

9. True remissions of long duration as well as long prodromic period, are peculiar to general paralysis of vesanic origin. The duration of this disease varies between 10, 12, or 15 years.

10. The doctrine of partial responsibility, far from seeming correct, appears to me as to be dreaded.

SHORTER NOTICES.

I. Diseases of the Bladder and Urethra in Women. By Alexander J. C. Skene, M. D. New York: William Wood & Co., 1878. 374 pages. Chicago: Jansen, McClurg & Co.

II. A Manual of Physical Diagnosis. By Francis Delafield, M. D., and Chas. F. Stillman, M. D. New York: William Wood & Co., 1878. Chicago: Jansen, McClurg & Co.

III. A Manual of Prescription Writing. With a full Explanation of the Methods of Correctly Writing Prescriptions; A Table of Doses, expressed in both the Apothecaries' and Metric Systems; Rules for Avoiding Incompatibilities and for Combining Medicines. By Matthew D. Mann. New York: G. P. Putnam's Sons. 155 pages. Chicago: Jansen, McClurg & Co.

IV. The Annual Report of the Board of Regents of the Smithsonian Institution. Showing the Operations, Expenditures, and Condition of the Institution for the year 1877. Washington, 1878.

V. Transactions of the Pathological Society of Philadelphia. Volume Seventh. Containing the Report of the Proceedings for the Sessions from September, 1876, to July, 1877. Philadelphia, 1878. Printed for the Society by J. B. Lippincott & Co. Chicago: Jansen, McClurg & Co.

VI. Transactions of the Medical Association of the State of Alabama. The Report of the State Board of Health. Thirty-first Session, 1878. Montgomery, Alabama, 1878. 315 pages.

Transactions of the Medical Association of Georgia. Twenty-ninth Annual Session. Atlanta, April 17, 18, 19, 1878. Atlanta, 1878. 279 pages.

Transactions of the Twenty-eighth Anniversary Meeting of the Illinois State Medical Society. Held in the City of Springfield, May 21, 22, 1878. Chicago, 1878. 275 pages.

VII. Lectures on Bright's Disease of the Kidneys. Delivered at the School of Medicine, of Paris, by J. M. Charcot. Collected and Published by Drs. Bourneville and Sevestre, Editors of the *Progrès Médical*, and Translated, with the permission of the Author, by Henry B. Millard, M. D., A. M. New York: William Wood & Co., 1878. 100 pages. Chicago: Jansen, McClurg & Co.

VIII. STUDIES IN PATHOLOGICAL ANATOMY. By Francis Delafield, M. D. Nos. 3 and 4, April and May, 1878. New York: William Wood & Co., 1878.

IX. HARVEY AND HIS DISCOVERY. By J. M. DaCosta, M. D., Professor of the Practice of Medicine, at the Jefferson Medical College, Philadelphia. Philadelphia, 1878: J. B. Lippincott & Co. Chicago: Jansen, McClurg & Co.

I. In this book Dr. Skene has done for diseases of the urethra and bladder in the female very much what Sir Henry Thompson has done for diseases of the urethra and bladder in the male. The book would imply, first, that disturbance in the female pelvis does not always proceed from lesion of the uterus, even though the uterus be involved; second, that symptoms which may not or may be referred to the uterus not uncommonly arise from lesions of other organs, and *vice versa.* And that in this view the urethra and bladder are worthy of much attention and study. Skene's endoscope (page 125) is an instrument with which the reviewer is familiar; this instrument unquestionably makes possible an accurate and easy inspection of the female urethra and bladder, and therefore supplies a want which hitherto has rendered impossible the intelligent diagnosis and treatment of many urethral and vesical diseases. We specially commend the chapter on vesico-urethral fissure, and the use of the endoscope in its diagnosis and management. The book is eminently practical and will prove an important aid, not only to the general practitioner but also to the specialist. The author is a pioneer in the American and English literature of the subject, and we gladly testify to the excellent work of so honest, intelligent and distinguished a gentleman. E. C. D.

II. This is an elegantly gotten up *resumé* of the principal points to be observed in the physical diagnosis of the diseases of the intra-thoracic organs. The illustrations are very fine, the plan of superimposed plates being adopted, with a lithograph on transparent tracing muslin, representing the external surface of the body, over all. This makes the plates a sort of anatomical dissecting model, and adds a kind of object teaching to the text. The blank interleaves are intended for the record of the owner's own observations, the work being intended as a sort of skeleton to be filled up from experience. We would cordially recommend it as a very valuable aid to the student and practitioner.

III. The instruction of this little book will be of advantage to many who have long felt the need of it. The art of prescription, for it is very much of an art, has been left to be learned by practical experience by most of our medical schools, and its acquisition is often a comparatively late accomplishment of the practitioner. It would be still more the case were the practice followed here of writing the directions in Latin generally followed in this country, as our author here states to be the case. We believe that there are comparatively few medical men in this country, and hardly any in some parts of it, who follow this plan. Still the directions here given for composing the kind of Latin used in prescription-writing will not be amiss, and so far as they relate to correct Latinity in the names of drugs are very useful. We see almost daily in medical journals prescriptions that would make a school-boy blush ; they are so ungrammatical and macaronic.

Such a book as this is always timely and should be in the hands of the profession generally.

IV. Of all the government documents, the Smithsonian Reports are perhaps at once the most popular and scientific, and we can say of them that there are very few series of volumes that are more worthy of a place in a general library. They give not only an account of the operations of the Institution for the year of issue, and which includes the history of a very large number of scientific investigations, but contain also scientific memoirs and translations, many of which are of the highest value, and cannot be found elsewhere in our language. In the present report we have, among others, a translation, occupying seventy pages, of Holmgren's memoir on Color Blindness in its Relation to Accidents by Rail and Sea, an article that, from its practical bearing, has elicited much attention in Europe, and which should be read by every railroad superintendent and steamboat inspector in the country. It is not necessary to notice the other memoirs in the volume ; though equally valuable in their way, they do not from the nature of their subjects come within the scope of this journal. The Report as a whole worthily keeps up the deserved reputation of the series.

V. The Transactions of the Pathological Society of Philadelphia form an elegant volume of one hundred and seventy-five pages. Of the papers it contains, two only relate specially to the nervous system: one a general paper on the Anatomy of the Cerebrum, by Dr. Harrison Allen, and the other a very fully reported case of Cerebral Abscess, by Dr. J. H. Hutchinson. Both are good articles; in the first, however, we notice an undue number of typographical errors, which ought to have been avoided. The other articles in the volume are in the main useful additions to pathological literature, and the volume as a whole is extremely creditable to the society and the publishers.

VI. The three volumes of Transactions of State Medical Societies in our rubric are fair specimens of their class. They contain the usual lists of members, reports of proceedings, addresses, and finally the reports and separate memoirs, which vary all the way between very good and comparatively bad, or at least unworthy of publication. Many writers do not care to bury their contributions in a society's transactions, and editors of such volumes do not have the discretion allowed them of rejecting articles that come into their hands; the society as a whole is ever ready to afford the compliment of ordering the publication of any paper that has been read before them ; indeed, we are not sure that the reading, according to the usage in some societies, insures the publication unless it is ordered otherwise by vote, and this invidious distinction is, of course, a very infrequent occurrence. These conditions react on each other, and the result too often is that a few meritorious contributions to medical literature are smothered in a mass of parliamentary proceedings, compilations and perfunctory reports and addresses, and the sum of them all is a volume that is seldom looked into and hardly ever referred to. We do not think that we are unduly severe ; it would, it seems to us, be better to have the publication of volumes of Transactions limited to special societies with restricted membership, in which it is understood that each paper shall undergo a rigid censorship before publication, and the more ephemeral literature relegated to the periodical medical press. If the American Medical Association, even, supported an official organ as does the similar British society, it would in our opinion be a preferable course to that adopted at present, of issuing a volume of Transactions.

We do not intend to say that the present volumes are not what they should be ; they are all that can be demanded or expected under present circumstances. The three reports that form the subject of the present notice contain very much valuable matter which, however, would in our opinion find a larger circle of readers, and fulfill a better function of usefulness if given to the world in the periodical medical literature of the day.

In the Illinois volume are several papers of interest as relating to the disorders of the nervous system. The first of these, by Dr. Earle, on exophthalmic goitre, is especially noteworthy in that the author has endeavored to elicit the opinions of medical men in various parts of the State as to its prevalence and frequency. The answers to his inquiries show that it is either very infrequent, or that it is very often unrecognized ; we think the latter is more likely the case. It is a disorder that, in many cases, may be successfully treated without recognition, the symptoms indicating the measures that should be adopted. The other papers are that by Dr. Hunt on the belladonna treatment of pertussis, and a short one by Dr. L. H. Corr on cholera infantum as a neurosis. The other reports are for the most part valuable, and the volume is, as a whole, creditable to the society.

In the Georgia and Alabama Transactions we have several gynecological and other papers of interest, and two or three on neurological subjects, which though well written and judicious, do not call for notice as containing anything especially novel. They, like the Illinois volume, are good specimens of their class.

VII. A work like this, coming to us with the authority of one of the great clinical teachers of Paris, needs no special endorsement on our part. The reader will find it a very concise, though comprehensive, and intelligible account of the pathology and clinical features of the class of disorders that, incorrectly as it appears to us, are commonly known under the general designation of "Bright's Disease." M. Charcot recognizes three disorders of the kidney under this appellation, confining himself solely to the consideration of the subacute or chronic forms; they are parenchymatous nephritis, or large white kidney; interstitial nephritis, or contracted kidney; and amyloid kidney. The acute forms of nephritis, with the exception of that sequent on scarlatina, are left unconsidered. The treatment of all forms of these affections is also not given in this work.

The translator and American editor has apparently done his work well, and the publisher has gotten up the book in excellent style.

VIII. We had inadvertently omitted to give these issues their due notice in their proper place, and now make amends. All we can say is, that they are worthy of a place in any physician's library; the illustrations are elegant, and the explanatory text is ample. We trust that the series will be continued.

IX. This volume, as stated in the preface, consists of an address delivered at the opening of the session of the Jefferson Medical College, with some illustrative notes. It is a very pleasantly written essay, showing considerable literary research, and will afford the reader better, perhaps, than he could otherwise obtain, a picture of the life and surroundings of the eminent physiologist. The rival claims to the discovery of the circulation of the blood are alluded to, but no very lengthy argument is offered to overthrow them; in fact, the title of Harvey to this discovery cannot well be gainsaid. The subject has been ably argued elsewhere. The make up of the book is excellent.

Editorial Department.

WE learn that an attempt is to be made to secure legislative consent to the appointment of a Pathologist to the asylums for the insane in the State of Illinois. We also learn that our esteemed contributor, Dr. H. D. Schmidt, of New Orleans, is an applicant for the position, if created.

While we have grave doubts, under existing circumstances, of the utility and expediency of making such an appointment by law, as is proposed, we feel bound to say, that if the office is created and can be filled by Dr. Schmidt, his appointment would do about as much as could be done in that way toward reconciling us to the proposed step on the part of the State. There is no man in this country better fitted, by honest, practical labor in the microscopy of the nervous system, to fill such a position than is Dr. Schmidt.

To make such an office really of value, it is required, first, that critical clinical histories of the cases should be made by those who are well versed in the recent anatomy and physiology of the nervous system, as well as with the vexed questions in the physiology and pathology of the nervous system, disputed points in diagnosis, and the characteristics of clinical groups and species. From such a stand-point and with reasonable thoroughness in the use of modern methods of research, the *ante-mortem* histories should be made of such cases as are to be brought to an autopsy. Then, on the supposition that the gross *post-mortem* examination has been carefully made, the microscopic examinations of the nervous tissues must be made by a candid, cautious, thoroughly experienced observer, not only practiced in microscopic technique, but familiar with the appearances of healthy nerve tissue, and with morbid appearances under the microscope, otherwise the observation of the microscopic pathologist will be either useless or worse than useless—misleading. But in the appointment of Dr.

Schmidt the office could be made a useful one, so far as the minute anatomy of the nervous tissues is concerned, since he appears to have about every requisite for filling such a position with profit. Everything will depend on the man who fills the office.

WITH the present issue commences the sixth volume of this journal; the fourth under its present title of the JOURNAL OF NERVOUS AND MENTAL DISEASE. The five years of its existence have been perhaps the most trying period of financial stress in the history of our country, and the fact that the JOURNAL has survived them is the best promise for its future continuance during the better period of which we trust we are at the threshold.

It was commenced to meet what was felt to be a want of the profession in this country, to establish a scientific medical periodical which should give to its readers a summary of the best results of investigation in the not narrow specialty of neurology from all parts of the world, and which should afford a medium for the publication of original work in this line in our own country. It was not expected that this latter would be its principal function, and for several years selections and translations filled a large portion of the first department of the JOURNAL. And though we now have no lack of high class original matter, we shall yet retain a place for translations of articles that may be of special or exceptional importance or merit, and not generally within the reach of the profession in this country. We are thankful to be able to say, however, that we have the prospects of making the first department of our journal more valuable and interesting than in any preceding period.

The second department, that of reviews, will be sustained as heretofore, but we shall have additional facilities and abler assistance not previously at our command. Dr. Meredith Clymer, of New York, it is hoped, will take special charge of the literature of insanity. As has been our previous custom, all articles in this department not written by the editors will be over the names or the recognizable initials of their authors.

The editorial department will be continued as affording a place for the discussion of general questions and such subjects as do not find their place elsewhere in the JOURNAL.

The Periscope will be kept up as hitherto, and it is intended that it shall, more fully than before, reflect the progress of neurological science. In it we shall hereafter give full abstracts and even complete translations of many important articles in foreign medical literature that are of interest to our readers.

As to any future plans of the JOURNAL, we have no changes to propose or new promises to make at present. But we will earnestly consider before the issue of another volume, whether some change in time of issue, and of make-up in other respects, will not render it more useful to the profession, and better suited to its needs.

OUR readers are doubtless all aware of the fact that Dr. Brown-Sequard now fills the chair in the *Collège de France*, made vacant by the death of Claude Bernard. On the second of December last he gave the opening lecture of his first course in his new position, and in which he laid down his programme for the year. It will perhaps not be a matter for surprise to many to know that it is to be devoted to directly combating almost every doctrine at present held in regard to the physiology of the brain, with especial reference, as might have been expected, to cerebral localizations. From the statements made in his opening lecture, it does not appear that his course is to be largely experimental, but chiefly based on a consideration of such facts as he has already collected, and which he has, at least in part, referred to in his lectures in this country, Great Britain and France, the past few years, and with which our readers are to some extent already familiar. We shall watch with some interest his course, but have very moderate expectations that it will be the means of establishing on a firm basis his peculiar doctrines, though his present position will give him the opportunity and the means for developing and placing them in their best light.

It is generally known to the medical profession and those interested in bibliography that Dr. John S. Billings, surgeon U. S. A., in charge of the National Medical Library at Washington, is now ready to print his great "National Catalogue of Medical Literature," as soon as Congress grants an appropriation for the purpose. This indexes under subjects, and by authors, books, pamphlets and original papers in nearly all the medical periodicals of the world; including over 400,000 subject entries, and making ten volumes royal 8vo of 1,000 pages each. This will be of the greatest value to physicians the world over, as it enables them to find analogues for peculiar and difficult cases, and thus often to save lives. In continuation of this work, it is now proposed to publish monthly, under the editorship of Dr. Billings and of his assistant, Dr. Robert Fletcher, M. R. C. S., a current medical bibliography under the title of the *Index Medicus*. It will be issued by F. Leypoldt, the bibliographic publisher, 37 Park Row, New York, at $3 per year, and will enter all medical books and index the leading medical journals and transactions in English and other languages. A full list of the latter, numbering over 600, will form a part of the specimen number of the *Index*, soon to be issued.

Owing to the necessity of suppressing an article, at the author's instance, just as we were going to press, this issue is not as large as we expected it to be, by several pages. In amends we shall try to have the space more than made up in the succeeding issues.

In this issue will be found the title page of the last volume, which was left out in the October number.

Periscope.

a.—ANATOMY AND PHYSIOLOGY OF THE NERVOUS SYSTEM.

THE FUNCTIONS OF THE BRAIN.—The following are the conclusions of a lengthy memoir by Luciani and Tamburini in the *Rivista Sperimentale:*

1. The excitable area of the cerebral cortex presents in dogs and cats, as well as in apes, considerable differences of disposition and boundaries, not only between different animals, but between the two hemispheres of the same animal.

2. We can establish in the dog by electric irritation, two distinct centres in the sigmoid gyrus for opposed movements for both anterior and posterior limbs.

3. In apes the excitable area for the members and the face is not limited solely to the ascending frontal convolution, as Hitzig claims, but extends also to the ascending parietal, the angular gyrus, etc., as Ferrier has shown.

4. The existence of a special epileptogenic zone in a fixed and definite cortical area is inadmissible, but, under proper conditions, the excitation in any excitable area whatever may cause an epileptic attack, which commonly begins in convulsions limited to the groups of muscles from the centres for which emanates the irritation.

5. The different excitable areas seem variously scattered, probably on account of their being endowed with different degrees of excitability. This may be presumed for many reasons, and especially that to produce a complete epileptic attack, acting on the same, requires a different intensity of the electric current.

6. It is absolutely impossible that the movements produced by electrization of the cortex can be due to a diffusion of the current to the dura mater.

7. The hypothesis that the movements produced by electrization of the cortical zone may be of reflex nature, in that there are in the excitable area so many sensory centres for the different parts of the body, does not stand against the facts of the decomposition, co-ordination to an end, and constancy of these, since the movements produced reflexly by peripheral excitation do not ever have such characters.

8. The phenomena of lost motility produced by ablation of the excitable zone, are of an absolutely paralytic, not ataxic nature.

9. The aforesaid paralytic phenomena are transitory, but they last the longer, that is the compensation takes place the more slowly, as we ascend in the animal scale.

10. To explain the recovery of the paralytic phenomena, the idea is inadmissible of a functional substitution of a neighboring area, or of the opposite hemisphere, nor can we say that the psycho-motor function is located in

the basal centres, but it is needful to admit that these last may physiologically be centres of voluntary motion, since the development or perfection of this their function produces the cure of the paralytic phenomena, and produces it all the more quickly the more developed this function is normally.

A. Pitres, *Thèse de Doctorat*, Paris, 1878 (abst. in *Revue des Sciences Médicales*).

To show that lesions of the subcortical white substance produce exactly the same effects as those of the convolutions, is the object of this important memoir. In order to fulfill it, M. Pitres insists in his first part on the possibility of subdividing and describing this apparently homogeneous *centrum ovale*, and first he divides it into three zones: prefrontal or anterior, occipito-sphenoidal, and fronto-parietal. This last zone, according to Pitres, is limited by two vertical planes: the one passing within five centimetres of the fissure of Rolando, the other passing one centimetre in front of the internal perpendicular fissure. Situated between the opto-striate bodies and the alleged motor convolutions, this median zone of the *centrum ovale* may be subdivided into four regions: pediculo-frontal, frontal, parietal and pediculo-parietal, each corresponding to four parallel vertical planes, carried to the foot of the three frontal convolutions to the frontal, the ascending parietal and the foot of the parietal lobules. Finally, each of these regions may be considered as itself formed of numerous bundles; thus in the pediculo-frontal we distinguish three: the superior, median and inferior.

This artificial limitation permits M. Pitres to classify the lesions of the *centrum ovale*, by comparing them to the superjacent convolutions; and in a second part he is then able to usefully analyze the numerous clinical observations. He first shows that lesions of the prefrontal zone, like those of the occipital, never cause any disorder, either motor or sensory, at least not directly, in the absence of complications. Lesions of the fronto-parietal zone alone, have, in the majority of cases, produced hemiplegic motor disorders, and sometimes also monoplegias. The existence of these monoplegias from injury to the *centrum ovale*, of numerous cases of aphonia from destruction of the left lower pediculo-frontal bundles, of brachial monoplegia from lesion of the upper and middle frontal bundles, etc., prove that the anatomical dissociation made by M. Pitres is true also functionally. The white substance of the centrum, in spite of its apparent homogeniety, is formed of bundles, each adapted to special conduction; in the fronto-parietal zone, the sole conductor of movements, the white bundles of fibres remain functionally distinct like the convolutions from whence they come; in fine, the lesions of the centrum and those of the superjacent convolutions produce exactly the same disorders.

It is to establish this last conclusion that M. Pitres gives the third part of his memoir. He shows that lesions of the centrum, like those of the fronto-parietal convolutions, are often latent; that the paralyses are often transient and incomplete, or progressive; that they are often preceded or accompanied by contractures, or even by epileptic convulsions; that, finally, they may be followed by slow contracture with secondary degeneration. He even discusses secondarily, the pathogeny of primary contractures, that of latent lesions, etc.

From this memoir leads a conclusion on which the author, perhaps, does not dwell with sufficient stress: the lesions of the *centrum ovale* and of the convolutions having the same symptomatology. Ought we not to admit with M. Vulpian, that the disorders of cortical origin are due to an irritation of the white fibres of the centrum, expanded in the convolutions, and not on the functionally homogeneous superjacent grey substance?

CHEMISTRY OF THE LIVING FROG'S HEART.—An article on the above subject by Dr. I. Gaule (*Archiv f. Anat. & Phys.*, 1878, III. & IV., p. 291), can be considered, unhesitatingly, as the most important contribution to muscular physiology which has appeared for years. His experiments (performed in the Strasburg Laboratory) refer to the chemical changes which occur in the excised frog's heart during its contractions. The entire organ was attached to Kronecker's double canula, through which the different fluids employed were passed. During observation the canula communicated with a small mercury-manometer, which registered the contractions on a kymographic cylinder. Each experiment was commenced by passing a current of 0.6 per cent. NaCl. solution through the heart, which as previous observers have found puts the heart into a state of complete diastolic rest, so soon as the last traces of blood are washed away. The results of Gaule's researches are sufficiently precise to be arranged in the form of theorems: 1. A heart which has ceased to contract after a thorough irrigation with 0.6 per cent. NaCl. solution, is recalled into activity by adding to the fluid a trace of sodic hydrate. The most powerful stimulation is obtained by a NaCl. solution containing one part of HNaO. in 20,000. Stronger solutions kill the heart, weaker fluids, down to 1 in 200,000, exert a less stimulant influence. The number of contractions in a given time does not vary, but the height of the contractions increases with the concentration, as is shown by the following table :

One part of HNaO. in 200,000 raised the mercury in the manometer in the average contraction, 3.5mm.

100,000	6.75
50,000	10.
20,000	16.75.

2. When the heart is once charged with a 0.6 per cent. NaCl. solution, containing a given amount of HNaO., the fatigue of the organ manifests itself in the gradual diminution of the hight of the contractions. On refilling the heart with a fresh quantity of this same solution, the strength of the muscle is restored to a certain extent. After a time, however, the heart is completely exhausted and does not respond to a renewed filling. The time requisite varies with each heart, but it is shortened by passing a large quantity of fluid through the heart between each two observations. If, however, the heart is again charged with the fluid, *which has once passed through it*, it is slightly revived. Hence the conclusion, that the work of the heart depends on the potential energy of stored up material, which is dissolved and washed out by the solution passed through the heart.

3. Considerable differences exist in the energy of different hearts, both as to the frequency and force of the contractions. This difference is traceable to the temperature to which the animal was subjected during life. The heart of frogs living in a cold space is always feeble, while the same organ of animals which have been kept in a warm room for several days displays uniformly considerable energy. The amount of energy of a heart depends on the quantity of "combustible" material, since the power of the heart of a cold frog can be raised to the standard of a "warm" frog by feeding the organ with the filtered alkaline extract of a "warm" heart. *The heart can be fed with the material from which the potential energy is derived, in the same manner as an engine is supplied with fuel, and as in the latter case the work done depends on the consumption of fuel.*

4. The filtered extract of the frog's heart contains salts, carbohydrates and albuminoids. The energy, however, of a heart fed with the usual solution of NaCl. and HNaO. containing traces of other salts or carbohydrates (grape-sugar, dextrine, glycogen), is not sensibly increased. Unable to obtain albuminoids free from salts, Gaule employed instead peptone, which was added to the usual solution. This was found to be fully as active as the extract of a "warm" heart. A heart completely exhausted by continued current fluid passed through it, could be again put into a state of maximal energy by feeding it with a solution containing 0.6 per cent. of NaCl., 1-200 per cent. of HNaO. and a trace of pure peptone.

5. On testing the alkalinity of a solution which had remained in the cardiac cavity until the cessation of contractility, it was found, that about one-half of the HNaO. had become neutralized. This was due for the greater part to carbonic acid. A small portion, however, of the alkali was combined with an unknown fixed acid. Different hearts furnished during the persistence of their contractions from 0.40 to 0.80 ccm. carbonic acid. A heart, on the other hand, which does not perform any contractions while charged with NaCl. solution, produces only about one-thirtieth of this quantity of carbonic acid during the same time. Hence the positive conclusion that carbonic acid is produced in the chemical changes accompanying contraction.

From the data furnished by his experiments Gaule calculates, that at least 8 per cent. of the liberated energy of the heart is utilized as mechanical work, while the rest disappears as heat.

In the same journal (p. 263), Dr. Stienor records similar experiments performed in Ludwig's Laboratory (Leipsic). The paper, although very painstaking, is by no means as conclusive as the previous one, but as far as it goes, it fully concurs with Gaule. His conclusions are, that the maintenance of cardiac contractility depends on the presence of a soluble organic matter, probably an albuminoid, while the actual occurrence of contractions demands the stimulus of an alkali. In his experiments the best results were obtained from the presence of 0.5 to 0.1 per cent. of sodic carbonate (which, however, as his figures show, is less stimulating than the sodic hydrate used by Gaule).

H. G.

THE SENSATIONS OF LIGHT AND COLOR.—A. Charpentier, *C. R. Acad. des Sciences*, March 20 and 27, 1878 (abstr. in *Revue des Sciences Médicales*). The sensations of light and those of color are the results of two very distinct functions, which while intimately associated in the habitual exercise of vision, can be neatly isolated in physiological analysis, the sensation of color being essentially variable, according to the point of the retina considered, and by numerous experimental conditions, independently of the luminous sensibility, but, reciprocally, the luminous sensibility may change under certain conditions while the sensibility to colors remains constant. Thus the eye that has rested in darkness possesses a much greater luminous sensibility than that which has been in constant activity, while in the one as in the other we find the same minimum for the appreciation of each color, *i. e.*, the chromatic sensibility is not modified either by exercise or repose. To explain the effect of resting the eye, on the sensibility to light, we may invoke the facts noticed by Boll, viz., that there exists in the retina a chemical substance of a red color, which the light bleaches, and which regenerates itself in darkness, and admitting next that the optic nerve is excited, not directly by the light but indirectly by the chemical modification which the light produces in the retinal red ; the resting of the eye in obscurity produces therefore an increase of sensibility to light on account of the presence in it of the photo-chemical red substance.

This hypothesis is confirmed by the following experiment : when we present to an eye that has just had its repose in obscurity, a pure color, that eye does not see a saturated color, but one strongly mixed with white ; thus we add to the chromatic impression a strong impression of white light, such as we obtain by the aid of the mixture of color and white in the rotating disks of Chevreul.

HERING'S THEORY OF COLOR.—A work recently issued, presents Hering's theory of the perception of color. It appears to be in antagonism to Young's theory, which assumes that three different sets of fibrils in the retina are susceptible to different wave-lengths of light. Hering assumes that the perception of color is the consequence of a chemical process: in general that when an assimilation is taking place, black is perceived by one retinal substance ; if dissimilation is taking place, white is perceived by the same substance. A second retinal substance furnishes the sensations blue and yellow, according as assimilation or dissimilation is taking place ; a third gives under corresponding conditions the sensations red and green. The white substance is also capable to some extent of being acted on by colored light ; the sensation of course still remains white. In this view, the process of perception of complementary colors is an antagonistic one. Thus, yellow and blue, which if mixed produce white, if perceived together will neutralize each other, leaving only so much of the white sensation as is due to the action of colored light on the white substance of the retina. This part of the theory, Professor Rood has tested by experiment. He finds that if a certain amount of yellow is mingled with a certain amount of blue, the white product has a total of as much light as the two colors had separately. But, if Hering's theory were correct, a certain amount of light ought to be extinguished when the colors are united. Professor Rood regards his experiments as fatal to the theory.—*Paper read before National Academy of Sciences* in N. Y., Nov., 1878. (Abstr. in *Science News*, Nov. 15.)

LOCAL VASO-MOTOR CENTRES.—At the session of the Soc. de Biologie, Nov. 9, 1878 (reported in *Le Progrès Médical*), M. Franck communicated the results of researches undertaken by himself on the peripheral circulations of the carotid, the vertebral, and the femoral arteries. Cl. Bernard was the first to observe the independence of certain local circulations under vaso-motor influence. In his experiments, M. Franck, having tied the artery, the circulation of which he wished to study, applied a registering manometer to its peripheral portion. Thus operating he was able to ascertain the variations of pressure. These, he observed, kept up even after he divided the vascular nerves of the region involved, *i. e.*, the superior ganglion of the sympathetic for the carotid. He concludes that the independent local circulations that exist for the majority of the organs of the body are regulated by peripheral nervous apparatuses. According to him, there is a complete independence between the carotid and vertebral arteries.

THE MODE OF NERVE-TERMINATION IN THE TACT-CORPUSCLES.—At the session of the Soc. de Biologie, Nov. 9 (rep. in *Progrès Médical*), M. Renaut gave an account of the intimate structure of the tact-corpuscles, as he had been able to develop it by the use of osmic acid. He described in the Meissner corpuscles a number of compartments built up out of connective tissue. In the majority of these are found cellular elements; and the nerve terminates in one of the central compartments. M. Renaut compared this arrangement to that of the dorsal cord in the external membrane of the embryo.

SWEAT-NERVES.—As the result of continued experimentation, F. Nawrocki (*Centralblatt*, No. 40, 1878) states, that the amount of perspiration varies as much in cats as it does in man. Some animals sweat easily, others scarcely at all or only in some, but not all parts (extremities). The sweat-nerves going to the fore paw of the cat pass from the cord into the anterior root of the fourth dorsal nerve, thence through the stellate ganglion of the sympathetic into the ulnar and median nerves. Excitation of any part of this tract starts the sweat gland of the fore paw into activity. On the other hand extirpation of the stellate ganglion leads to degeneration of the sweat-nerves in their peripheral course, and hence stimulation of the ulnar or median nerves will now have no longer any influence on the sweat glands. The sweat-nerves of the hind paw pass from the cord with the anterior roots of the last (13th) dorsal and first and second lumbar nerves, enter the abdominal sympathetic and thence the sciatic nerve.

The statements of Luchsinger that minor sweat centres exist throughout the cord, the author could not confirm with much precision. Out of a large number of experiments he succeeded only three times in obtaining secretion of sweat after transverse section of the upper part of the spinal cord asphyxiating the cats. All attempts to obtain perspiration by stimulating sensory nerves after separation of the cord from the medulla failed.

THE SECRETORY NERVES.—Communication by M. Vulpian to the Acad. des Sciences, Sept. 9 (rep. in *Bull. Gén. de Thérapeutique*).

I remarked at a late *séance* the following fact observed by Luchsinger: that if we divide the sciatic nerves in a cat, the chloro-hydrate of pilocarpine, injected under the skin on the same day as the section, causes an abundant secretion of sweat on the digital pulps of the corresponding members as well as in the other limbs; but if the section has been made more than six days, the injection of the pilocarpine has no sudorific effect on the digits of the corresponding foot.

Is the case the same with the sub-maxillary salivary gland, the excito-secretory nerves of which are well known, and on which jaborandi and its alkaloid, pilocarpine, act as on the sudoriparous glands? This is the question to the investigation of which I applied myself. My experiments were performed on dogs.

Researches, under my direction, by MM. Carville and Bochefontaine, have demonstrated that jaborandi still excites the secretion of the sub-maxillary gland of the dog, when we inject the infusion of this plant into a vein, the dog on which the operation either of dividing the chorda tympani, or of the cervical sympathetic, or after excitation of the superior cervical ganglion, or even after in the same dog, we have removed the cervical ganglion and cut the lingual nerve joined to the chorda tympani.

On April 26, I examined the action of jaborandi on a dog that had undergone on the 19th, that is, seven days previously, the section of the lingual nerve of the right side, above the point where the chorda tympani leaves it to enter the sub-maxillary gland. As in all the other experiments which I describe in this communication, the investigation of the action of jaborandi was performed on a curarized animal undergoing artificial respiration. A metallic tube was fixed in each of Wharton's ducts. A small amount of rather strong infusion of jaborandi was injected into one of the saphenous veins towards the heart.

In the dog in question we found that the intra-venous injection of infusion of jaborandi caused almost immediately an abundant flow of saliva from Wharton's duct of the left side (*lingual intact*); on the right there was also a flow, but less abundant and appearing a little later on the left, and the saliva was more viscous and stringy on the right than on the left. When the excito-salivary action of the jaborandi was exhausted, a new injection was made, and again the same difference in the secretion of the two sides was observed.

Jaborandi, therefore, produces its ordinary effect on the sub-maxillary salivary gland seven days after the division of the chorda tympani; this action is only slightly enfeebled and the characters of the produced secretion are a little modified.

But the chorda tympani is not the only excito-secretory nerve of the sub-maxillary gland. The nerve fibres of the great sympathetic sent to that gland, contain, without doubt, freno-secretory fibres, but they are especially made up of excito-secretory fibres, as all experiments demonstrate.

To compare the effects produced, after several days, on the sub-maxillary gland, by section of the chorda tympani, and those caused, under the same conditions by the section of the glandular fibres from the sympathetic, it is needful to divide the cervical sympathetic or, better, to excise the superior cervical ganglion. This last operation was performed on a dog, on the right side, on July 21, and the action of jaborandi was examined on the 31st of July, that is, ten days after the operation. The two lingual nerves were cut transversely above the point where the secretory filament from the chorda tympani detaches itself; then we faradized successively the peripheral ends of these divided nerves at the point where they still contained the whole of the chorda tympani, and found that the secretion of saliva was more free on the left than on the right side (where the sympathetic ganglion had been excised). This result was repeated a number of times; we injected into the saphenous vein some centimetres of infusion of jaborandi: the saliva flowed almost simultaneously from the two Wharton's ducts; the quantity was rather greater on the right side than on the left.

We see, therefore, that jaborandi still exerts its excito-secretory action on the sub-maxillary gland some days after the excision of the superior cervical ganglion; a ganglion from which seem to arise all or nearly all the sympathetic fibres innervating the sub-maxillary gland.

But the comparison cannot be made between the effects observed in the cat after section of the sciatic or the nerves of the brachial plexus on the sweat glands of the digital pulps of that animal, and those that section of the secretory nerves produces on the function of the salivary glands in dogs, unless we cut in the latter case, in the same animal, the chorda tympani and the sympathetic secretory fibres.

This experiment was performed on three dogs:

In one of them the lingual and sympathetic of the right side were cut on the 30th of April, 1878. On the 8th of May a little infusion of jaborandi was injected, towards the heart, in one of the sapheni veins. Some instants later the saliva began to flow from the duct of Wharton drop by drop, the flow lasting several minutes, then slackening, but revived again by another injection. (Wharton's duct was prepared only on the side of the cut nerves.)

In a second dog the section of the two nerves was made on the 19th of June, 1878. On the 3d of July we injected into one of the saphenous veins a small quantity of infusion of jaborandi. Almost at once the saliva began to flow from Wharton's ducts on both sides, but more abundantly and rapidly on the side of the intact than on that of the divided nerves.

In a third dog, the lingual joined to the chorda tympani was divided, on the right side on July 5th, 1878, and on the same day the superior cervical ganglion was completely excised. On July 18th some cubic centimetres of jaborandi infusion were injected towards the heart, into one of the saphenous veins. As in the former experiments a flow of saliva from both ducts of Wharton, more abundant and appearing more quickly on the left (nerves intact) side than on the right.

Having sufficiently verified these results, it is yet needful, before concluding, to assure ourselves that the sub-maxillary salivary glands do not receive other secretory nerve fibres than those derived from the chorda tympani and the superior portion of the cervical sympathetic. To ascertain the facts as to this I faradized energetically the superior extremity of one of the sciatic nerves, in a dog that had undergone, ten days previously, the section of the chorda tympani and the excision of the superior cervical ganglion on the right side. Under the influence of this excitation an abundant flow of saliva was produced from Wharton's duct on the left side, while not a single drop flowed from the tube in the duct of the opposite side.

It follows, therefore, from these experiments, that section of the excito-secretory nerves of the sub-maxillary salivary gland does not exert such an influence on the function of the gland, as does the section of the sciatic over the sudoriparous glands of the digital pulps of the posterior limb. Jaborandi still acts on the sub-maxillary gland many days after the section of the excito-salivary nerves, while it or its alkaloid, pilocarpine, from the sixth day after the section of the sciatic (the nerve that appears to contain all the sweat exciting fibres for the hind limb), has no more action on the sudoriparous glands of the corresponding member.

What is the cause of such dissimilarity of the results of two experiments that offer at first sight so great a resemblance ? Is it due solely to the difference of constitution of the substance of the proper anatomical elements of the two kinds of glands, sudoriparous and salivary ? Shall we seek the reason in the circulatory modifications produced under the influence of the section of the nerves, which modify differently the functions of the simple glands (sweat glands) from those of the composite glands (sub-maxillary) ? or rather, shall we not attribute this dissimilarity to the enormous quantity of nerve cells distributed, either singly or in more or less voluminous ganglionary groups over the whole length of the secretory nerves destined for the sub-maxillary gland, cells which, after the division of these nerves apparently prevent the fibres from gradually losing their excitability to their peripheral terminations ?

The latter explanation seems most satisfactory, but new researches are needed to definitely establish its value.

THE INFLUENCE OF THE QUANTITY OF BLOOD IN THE MUSCLES ON THEIR IRRITABILITY.—The following is the *resumé* of a note by M. Schmoulewitsch to the Acad. des Sciences, Paris, in September last, as given in the *Bulletin Générale de Thérapeutique :*

The experiment of Stevson, dating from the seventeenth century, and which consists in producing a paralysis of the posterior members by a ligature of the abdominal aorta, proves the intimate relation between the circulation of blood in the muscles and their function. M. Brown-Séquard has shown that in animals and even in man, the stiffened muscles recover their contractility after injection of arterial blood. It may be admitted in a general way that bloodless muscles lose their irritability and cease to perform their functions.

In repeating these experiments, I have found that the muscles in becoming anæmic do not at once lose their irritability. On the contrary, this for a while is even increased, and only commences to disappear after a certain degree of augmentation. The same phenomenon is observed after section of a nerve, the irritability of the corresponding muscle is increased for the first few moments. This latter phenomenon should, in my opinion, be attributed also to anæmia, as this is the immediate result of nerve section.

The celebrated experiments of MM. Cl. Bernard, Vulpian, and others have demonstrated, that in the muscular nerves there are vaso-motor branches, the excitation of which produces a complete anæmia of the muscles, while their section causes a hyperæmia and increase of temperature. But we have, as a first result of the section, a mechanical excitation of the nerve.

That the anæmia is the cause of increased irritability of the muscles, I have proven by the following experiments :

1. After compression of the aorta or ligating the artery of a muscle, we cannot produce any augmentation of the irritability after section of a nerve. This demonstrates that this augmentation depends exclusively upon the circulation, for, as I say, the circulation once interrupted, the section of the nerve is without effect.

2. In curarizing an animal to the stage of complete paralysis, we always find an increase of muscular irritability following the section of the nerve. Here evidently, only the vaso-motor nerves can act, since, as has been demonstrated, they are not easily paralyzed by curare.

Anæmia, therefore, like certain affections of the nervous system that disorder the functions of the vaso motors, should increase the muscular irritability, a fact observed clinically, but not as yet sufficiently demonstrated theoretically.

TERMINATION OF NERVES IN THE VOLUNTARY MUSCLES.—At a recent session of the Acad. des Sciences, Paris (rep. in *L'Union Médicale*, Nov. 9), M. Tschiriew offered the following communication in regard to the nerve terminations in the striped muscles.

The termination of the nerves in the striped muscles has been the subject in recent times of numerous investigations, which, in spite of their interest, have not as yet given us complete light upon the subject. It has been thought, indeed, for example, that the terminations of the sensory nerves in the muscles have been found, but this discovery due to defective investigations cannot be considered as exact. Besides, all the efforts to find the intermediate forms between the terminations *en plaques* and the motor terminations in the frog have been so far without success.

"The procedure of staining the nerves with the chloride of gold, recently communicated by M. Ranvier, having supplied me with an excellent and certain method for the study of the nerve terminations, I undertook a series of researches that have yielded me some new results, which I have the honor to detail.

1. "The non-medullated nerve fibres found in the smooth muscles of the frog, such as the thoracic cutaneous muscle, and what have been considered hitherto as sensory fibres, do not appertain to the muscle, properly speaking, but to its aponeurosis. These fibres, coming from the intra-muscular nerves, form in the aponeurosis a large-meshed net-work. Their terminations are similar to the nerve terminations in the cornea.

"It is evident from their microscopic structure as well as from their anatomical relations, that these nerves of the aponeuroses ought to be considered as centripetal nerves from the muscle. The need of admitting the existence of these nerves has been demonstrated in a physiological memoir I recently published *on the origin and signification of the knee phenomenon and other analogous phenomena.*

"Nerve fibres similar to those, the existence of which in the frog I have noticed, have been detected also in the aponeuroses of other animals.

2. "I have found it quite impossible to determine in the dissociated muscles of the frog, and of some other species of animal (tortoise, frog, lizard, snake, and rabbit), the presence of non-medullated fibres, other than those that appertain to the vascular or aponeurotic nerves, and the presence of terminations other than motor terminations.

3. "I have, on the other hand, been able to find in many species of animals new forms of nerve terminations which constitute the intermediate forms between the motor terminations, such as we see in the frog, and the end plates. I have found these in the tortoise, the triton, the salamander, the lizard, and the serpent. In the three first named these are the only terminations I could find, while in the lizard and the serpent I met with them together with the end plates, especially in young muscular fibres.

"The simplest form of these terminations is seen in the tortoise; non-medullated nerve fibres ramify without anastomosing and terminate in the muscular bundles by stems that are sometimes smooth, but which are more frequently moniliform or surrounded by granulations strongly tinged by the gold. These granulations, which are disposed around the terminal stems, are sometimes so numerous that they form, as a whole, a resemblance to the terminal arborization of a little motor end plate.

"These new forms of nerve terminations all present this peculiarity, that they are only found in non-medullated nerves, although the latter always come from medullated nerves. In the snake the non-medullated fibres may run a long distance.

"In cases where the nerve ends in the muscle by a well developed plate, we never observe more than one such for each muscular fibre; when, on the other hand, we meet with the terminations I have described, we generally find many nerves ending on the same muscular fibre, and in the snake there may be as many as six or seven."

A more detailed memoir with illustrations will shortly be published.

INFLUENCE OF PHYSICAL AGENTS ON THE PERIPHERAL NERVES.—At the session of the Soc. de Biologie, Oct. 19 (rep. in *Gaz. des Hopitaux*), M. Onimus offered the following communication:

Mechanical excitations and thermic agents often give the same results as metallic applications in hysterical cases, that is to light currents of electricity. This shows very clearly that in all the modifications of hemianæsthesia in achromatopsy and contracture, we have principally to do with molecular changes in the peripheral nerves.

A simple shock, or more exactly, very frequently repeated and short vibrations may also cause the same modifications of the peripheral nerves in cases of hysteria. The lesion appears, therefore, to be solely a sort of benumbing of the conductibility of the peripheral nerves. These different agents come, so to speak, to the assistance of the nerve to force it to vibrate. As we have said in our article on *contracture*, there is for the nerves as for the muscles, an intermediate state between health and genuine disease, the sole lesion of which is slowness and defect of vibratory motility.

In the sensory nerves we have even more characteristic examples of these various impressions of the peripheral nerves, for thus a shock or blow struck on the forehead causes the impression of phosphorus, the same as a current of electricity. It is the same with the auditory sense in which electric currents produce particular noises, differing a little according to the nature of the current, but which resemble those due to other causes.

It is also very useful to compare these phenomena to those recently brought by M. Hirn to the attention of the Academie des Sciences. A bar of iron may be heated instantaneously more than 30° by the stroke of a hammer, and, at the expiration of a second, the former temperature reappears. It is not, it is true, a real increase of temperature, but an absolutely subjective phenomenon. The sonorous vibrations have, by their rapidity, shocked the cutaneous nerves so as to create an impression of heat.

These facts demonstrate that in all cases vibrations arising from different sources of motion, applied to the peripheral nerves, give rise to the same sensations; and in a subjective way all the excitants, electric currents, and especially those of tension, the vibrations of a musical instrument, of thermic agents may, when the nervous system is in a normal condition, produce analogous phenomena.

THE following are the titles of a few of the recent papers on the Anatomy and Physiology of the Nervous System, of which we have not yet given abstracts in the JOURNAL :

CARPENTER, The Effects of Attention on the Bodily Organs, *Brit. Med. Jour.*, Dec. 14, 1878.—HURD, The Physiology of Sleep, *Boston Med. and Surg. Journal*, Dec. 26.—CHARCOT and PITRES, New Contribution to the Study of Motor Localizations in the Cortex of the Cerebral Hemispheres, *Revue Mensuelle*, Nov.— TARCHANOFF, On the Psychomotor Centres in New-born Animals and their Development under Different Conditions, *Ibid.* —EULENBURG & GUTTMAN, Physiology and Pathology of the Sympathetic System of Nerves. Transl. by A. Napier, M. D., Part II., *Jour. of Mental Sci.*, Oct.—BUCKE, The Moral Nature and the Great Sympathetic, *Am. Jour. of Insanity*, Oct.

b.—PATHOLOGY OF THE NERVOUS SYSTEM AND MIND AND PATHOLOGICAL ANATOMY.

SYSTEMIC DISORDERS OF THE SPINAL CORD. Flechsig, *Archiv der Heilkunde*, XVIII., 288, 243, 1877; and XIX., 53, 1878. (Abstr. in *Revue des Sciences Médicales.*)

In the anatomical part of his memoir, Flechsig endeavors to give precisely the track of the pyramidal fibres, considered as forming a separate system as regards their functions, their origin, and their terminations. This chapter reproduces in part the results published by him in his work on the systematic description of the white substance of the nerve centres. Nevertheless, in carrying out his embryogenic researches, the author has modified his views regarding the higher origin of the pyramidal fibres. Formerly, he considered these fibres as terminating above in the central ganglia of the hemispheres, especially the lenticular nucleus.

At present Flechsig is convinced that the pyramidal fibres, which are the first to be provided with a medullary sheath in the embryo, do not have any connection with the central ganglia, but pass directly to the grey matter of the convolutions. If we follow their course from below upwards, we observe that at their exit from the pons, the pyramidal fibres pass in the third quarter of the lower portion of the peduncle (in going from without inwards). In the internal capsule, externally, they pass directly by the lenticular nucleus; internally they respond to the median portion of the optic thalamus, and more above to the nucleus caudatus, from which they are separated by a bundle of fibres going for the optic thalamus to the radiant crown. They penetrate into the centrum ovale at the level of the third posterior fourth of the nucleus caudatus, above the superior border of the lenticular nucleus. From this point they intermingle with foreign fibres in such a manner that their course cannot be followed far. It is, therefore, impossible to state precisely the parts of the grey cortex in which these fibres terminate.

The only thing that is certain is, that after their entry into the centrum ovale, these fibres spread out but very little, and that they preserve in great measure the direction that they took in the internal capsule; whence it may be presumed that they pass to the grey matter of the central convolutions and the frontal convolutions adjoining them.

The description of the intra-bulbar and intra-spinal track of these pyramidal fibres is only a repetition of that given by Flechsig in the work above cited. (See this journal for 1877, p. 360.)

Pathological Anatomy.—In the second part of his memoir, Flechsig engages in the demonstration that the secondary degenerations of the cord, consecutive to brain lesions, have their location in the pyramidal tracts, and that according to all appearances only the pyramidal fibres are involved to the exclusion of the foreign fibres intermingled with them.

The anatomico-pathological researches of Tuerck, Vulpian, and Charcot show that lesions of the grey cortex are not followed by secondary degenerations, unless they are situated in the grey matter of the central or the neighboring convolutions. The observations of Tuerck and Charcot show that the secondary degenerations of the pyramidal fibres are consecutive to lesions of the grey matter of the motor zone and the subjacent white substance. Moreover, the recent researches of Westphal have demonstrated that very superficial lesions of the grey substance, such as are found in progressive general paralysis, rather frequently give rise to secondary degenerations of the pyramidal bundles.

As to lesions involving the grey matter of the central ganglia of the hemispheres, the observations of Charcot have as yet not supplied the direct proofs that they can exist without giving rise to secondary degenerations. On the contrary, however, Tuerck has published the account of a case in which a *foyer* the size of a hazelnut existed in the caudate nucleus without any degeneration of the pyramidal fibres. In two other cases of Flechsig's, the external zone of the lenticular nucleus was destroyed and the pyramidal fibres were also intact. On his part Tuerck has reported two cases in which the upper portion of the optic thalamus was altered without secondary pyramidal fibre degenerations.

But we still lack direct proofs showing that lesions of the first and second members of the lenticular nucleus, the tails of the caudate nucleus and the lower posterior portion of the optic thalamus are incapable of producing this secondary degeneration.

Lesions of the internal capsule are those that most frequently give rise to secondary degenerations of the pyramids. Flechsig, however, considers as erroneous the opinion of Charcot that only those lesions that involve the two anterior thirds of the capsule can produce them. Thus in the two cases of Tuerck, mentioned above, the secondary degeneration involved the third posterior fourth of the internal capsule, and it is just at this horizon, according to Flechsig, that the pyramidal fibres coming from the cortex traverse the capsule. Moreover, in five observations of secondary degenerations of the pyramids that Flechsig himself reported, it was just this part that was involved.

In the cerebral peduncle the study of secondary degenerations is more complicated.

The study of the cerebrum of newly born individuals shows that the fibres extending from the peduncle to the internal capsule preserve their original relations in the latter. The fibres which are the most internal in the peduncle are the most anterior in the capsule, and *vice versa*.

Thus are explained the facts observed by Charcot and Vulpian: in case of lesion in the internal capsule, the more anterior it is in this tract, the nearer the secondary degeneration in the peduncle is to its internal border.

In the medulla and cord the degeneration always invades the pyramidal fibres as they have been described by Flechsig. The extent of the degeneration (as regards the number of fibres involved) depends on the dimension and locality of the primary lesion.

Indeed, at the horizon of the internal capsule, the section of the pyramidal bundle presents a surface fifty times smaller than that of the cortical motor zone in which these fibres terminate. A lesion of the same extent would therefore entrain the degeneration of a greater number of fibres, when it involves the third posterior fourth of the internal capsule, than when it occupies the motor zone of the convolutions.

The distance from the side of the cord the degeneration extends in the anterior and lateral columns, and its extent in length present as many variations as does the distribution of the different bundles of the pyramidal fibres in this portion of their course.

When the degeneration occupies two homologous columns of the cord at the same time, it may be consecutive to a bilateral lesion of the hemispheres, or to a lesion involving the cord itself.

Secondary degenerations of the pyramidal fibres appear to have no reaction on the grey nuclei of the medulla and on the grey matter of the anterior horns.

From all this, Flechsig concludes that the secondary degeneration of the pyramids constitute rather a lesion of an elementary system of fibres.

In a last article, he defends his method of investigation, based on embryogenic researches and the anatomical results obtained with its use, against the attacks upon it by Mayser. To the results of Flechsig Mayser had opposed, in a recent article (*Arch. f. Psychiatrie*, VII.) the results obtained by him in experiments of vivisections of new-born animals.

PRIMARY DEGENERATIONS OF THE PYRAMIDAL BUNDLES.—In a third article Flechsig undertakes to study the localization and the precise nature of the lesions met with in the spinal disorder described by Professor Charcot under the name of amyotrophic lateral sclerosis.

I. The lesions of amyotrophic sclerosis are essentially a primary and symmetrical lateral sclerosis of the white lateral columns, a destructive atrophy of a great number of multipolar cells in the anterior horns, and a degeneration of a considerable number of the motor fibres of the anterior roots. But Flechsig endeavors solely to show that these different elements thus involved belong all to one system, which represents the most direct route of transmission connecting the cortical motor centres with the peripheral muscles.

a. Thus the lesions involving the white substance of the cord will not be limited to the lateral columns taken in their *ensemble*, as Charcot would have it. According to Flechsig, of all the cases of amyotrophic lateral sclerosis collected by M. Charcot, only six can be utilized in solving this question of localization.

But, in analyzing these six observations, the German author comes to the conclusion that the lesion of the white substance always reaches its greatest intensity at the horizon of the pyramidal bundles, as he understands them. (See above.) In the majority of cases one part (the smallest) of the pyramidal bundles passes along the anterior columns, while the other forms the posterior segment of the lateral columns. These are the place of election of the lesion in amyotrophic lateral sclerosis, only because they usually contain the greater part of the pyramidal fibres. But the anterior columns are likewise involved in the majority of cases. If they are not always involved this is owing to the distribution of the pyramidal fibres in the cord. It may happen, indeed, that all the pyramidal fibres decussate in the medulla and pass down in the lateral column of the opposite side; in this case they are lacking in the anterior columns.

It is none the less true that, in amyotrophic sclerosis, the lesion rather frequently involves that part of the lateral columns that is not composed of the pyramidal fibres. In one case even the posterior columns (more especially the columns of Goll) were invaded.

Supporting himself on the preceding facts, Flechsig criticizes the name primary symmetrical lateral sclerosis. This name has the great inconvenience of losing sight of the fact that the only system constantly invaded is the system of the pyramidal fibres, which is habitually distributed in the anterior and lateral columns; and on the other hand the lesion, in lateral amyotrophic sclerosis, is not truly symmetrical except when the distribution of the pyramidal fibres in the cord is itself symmetrical, which is not always the case. Finally, he does not admit as yet demonstrated, that the lesion has always the white substance as its starting-point.

On these different accounts, he proposes to substitute for the term adopted by M. Charcot, the name primary fascicular sclerosis of the pyramidal bundles. We must, moreover, distinguish a simple and a complicated form, according as the anatomical lesion remains limited to the pyramidal bundles, or invades those adjoining.

b. The lesions of the grey substance consist in an atrophy with complete degeneration of the multipolar cells which we find in the anterior horns; that is to say, of the nervous elements that are in direct communication with the pyramidal fibres of the anterior lateral columns. Moreover, the nervous elements which in the medulla are, in a fashion, the homologues of the great motor cells of the anterior horns, seem to be the only ones there involved in cases of amyotrophic lateral sclerosis.

c. In the typical cases of amyotrophic lateral sclerosis, the lesion spares the posterior roots, and only invades those fibres of the anterior roots whose axis cylinders are merely the prolongations of the ganglion cells of the anterior horns. It consists in a degeneration of the nervous elements, the interstitial tissue remaining intact. Moreover, at different horizons the number of altered fibres appears to be in relation with the corresponding number of degenerated multipolar cells.

In fine, Flechsig comes to the conclusion that in a certain proportion of cases of amyotrophic lateral sclerosis, the lesion involves, sometimes exclusively, sometimes in a predominant way, a collection of nervous elements which maintain very intimate functional relations. These elements (pyramidal fibres, multipolar cells of the anterior horns, motor fibres of the anterior roots) constitute what Flechsig calls the *direct cortico-muscular system*, that is, the most direct route of transmission from the grey cortex of the convolutions to the motor cells of the anterior horns whence originate the motor fibres destined to the striated muscles. In fact, as has been said before in the analysis of the anatomical part of Flechsig's memoir, the pyramidal fibres, according to the author, extend directly from the peduncles to the cortex, without touching the central ganglia of the hemispheres.

II. In a special chapter the author discusses lengthily the histological character of the lesion of the white substance of the cord in amyotrophic sclerosis. He is disposed to admit that it is rather a primary degeneration of the nerve fibres, and not a primary hyperplasia of the interstitial connective tissue with secondarily atrophy of the compressed nerve elements. But in fact, direct proofs derived from microscopic examination are lacking, and Leyden claims that, in a proportion of cases of amyotrophic sclerosis, the lesion primarily affects the neuroglia, and that the atrophy of the nerve elements is only a secondary alteration.

In reality, according to Flechsig, the differences noticed by Leyden in various cases of amyotrophic sclerosis, are not more marked than those which are found in the comparison of a number of cases of secondary degeneration, some recent and others of older date.

Moreover, the systematic distribution of the lesion in the majority of cases of amyotrophic sclerosis, is a sufficient reason for holding that the primary trouble is in the nerve elements. Indeed, the different systems of fibres which by their union form columns of the cord, are nourished by a single vascular net-work, and the connective tissue which serves as a support to both vessels and nerve fibres, is alike in all. We do not see, therefore, says he, why a lesion having its point of departure in the interstitial connective tissue and limited to a small portion of the transverse section of the cord, should propagate itself through the whole length of the spinal axis without involving the whole of its white substance.

In closing, the author announces that in a later communication he proposes to define the precise *role* that the direct cortico-muscular transmission system plays in the amyotrophic lateral sclerosis of Charcot.

Partial Nerve-Section.—In the *Archiv f. Anat. und Phys.* (1878, III. and IV., p. 218), Dr. I. Steiner describes a method of splitting the vagus longitudinally (in rabbits). The best spot for the operation is the part immediately below the ganglion of the vagus, where the nerve is flattened. On testing the artificially separated bundles, it was found that the external bundle comprised only the sensory fibres, while the internal half consisted of the motor filaments innervating the heart, larynx and œsophagus.

Degeneration of Severed Nerves.—After a methodic study of the degeneration of the peripheral stump of the divided sciatic nerve (Guinea-pig) Guiseppe Colasanti (*Archiv f. Anat. und Phys.*, 1878, III. and IV., p. 206) claims that we must distinguish between the degeneration proper, which is not observable until after the lapse of 72 hours, and the traumatic change found in the peripheral stump, extending as far as the first annular constriction.

This change, quite marked within 24 hours after the division, consists in the optic disappearance of axis cylinder and medullary sheath. The interior of the neurilemma is filled with a homogeneous, strongly refracting mass, without apparent structure.

Colasanti's description of the degenerative process refers mainly to the behavior of the lately described medullary segments. About the fourth day these segments increase in width and become less refractive, until they have the appearance of paraffine. Hereupon the segments, formerly joined like cones, fitting into corresponding depressions, begin to separate, until the nerve seems to consist of a series of elongated drops. This finishes the first stage, ending about the sixth day.

As a second stage in the degeneration process, C. describes the occurrence of folds and prominences on the surface of these drops, leading to further segmentation. After the end of ten days the contents of the nerve consist of a detritus of finely granular substance, in which minute drops of fatty lustre, free nuclei and leucocytes are suspended.

OPHTHALMIC MIGRAINE.—The ocular phenomena of migraine are sufficiently prominent to be noticed by every one, but they have usually been looked on only as complications. We find in the *Gaz. des Hopitaux*, Oct. 5, an article on ophthalmic migraine as recognized by M. Galezowski. Many observations led this author to the conviction that many apparently very serious ocular troubles were purely of a nervous nature, and that they might alone constitute a true migrainous attack. Such, for example, was an attack of amblyopia, apparently indicating serious trouble, appearing all at once, after a violent nervous commotion, in a woman subject to frequent such headaches, and which disappeared altogether in the course of a few days. In another case a young man who was subject to very frequent and severe migrainous attacks, but who passed once several months without one, during which period he had, four to six times a week, amblyopic visual disorders, lasting twenty or thirty minutes each time. Such instances as these seem to indicate that the ordinary symptoms of migraine may be at times replaced by ocular disorders, purely neurotic in their nature, but closely simulating serious structural disease. This brings up another point: if this be the case, the diagnosis of ocular troubles may be embarrassed by the fact on the one hand, and on the other, there may be already existent organic disease of the eye to complicate the diagnosis of the neurosis. M. Galezowski has canvassed a vast number of observations in his experience, and has endeavored to establish the diagnosis of these special neuroses of the eye. He concludes that there is a special form of migraine, which he calls migraine of the eye, or ophthalmic migraine.

He recognizes four varieties of this affection: periodic hemiopia, scintillant scotoma, migrainous amaurosis, and photophobia. Of seventy-six cases collected forty-one were females and thirty-five males. It appears most frequently in those who have long been liable to the ordinary form of migraine, and especially in nervous hysterical women. Sometimes the ophthalmic symptoms are preceded by a violent headache, but, more frequently, they come on all at once without premonitions, and are then often but not always followed by pain in the head, nausea, and other usual characters of migraine. The visual disorder is characterized by dimness of vision, speedily followed either by a central scotoma, or by hemiopia. In the cases where the ordinary symptoms of migraine make their appearance, as they do frequently, they appear only after the cessation of the visual disorders. In some cases the usual form is complicated with sudden blindness, accompanied or not, with scintillations, etc.

According to M. Galezowski this ophthalmic migraine is a neurosis of one of the portions of the fifth nerve which supplies the vaso-motor filaments either to the central organs of vision, such as the tubercula quadrigemina, the geniculate bodies, and the chiasm, or to the peripheral parts, such as the optic nerves and retina. The irritation of these vaso-motor fibres, like that of the other branches of the fifth pair, will ordinarily produce pain. It may react only in the visual centres, producing hemiopia, or there may be a scintillant scotoma, with jagged lightning-like appearances, or the resemblance of a fortification-like polygon, due to irritation of the vaso-motor fibres of the retina. The photophobia and suffusion are readily explained by supposing a propagation of the irritation to the lachrymal gland and the ciliary nerves.

The treatment recommended by M. Galezowski for this form of migraine is the same as for the other forms, especially by acting on the digestive canal for a long period. By means of light saline purgatives he has been able to relieve both the head and eye symptoms. Bromide of camphor and bromohydrate of quinine seem to have a very good influence in these ocular crises.

Cerebellar Diseases.—H. Nothnagel, *Berliner klin. Wochenschr.*, No. 15, 1878 (abstr. in *Centralblatt f. d. med. Wissensch.*).

Diseases of the cerebellum often run their course latent, without regard to the kind of disorder or the size of the lesion. But this can only occur when the disease is located in one hemisphere, since the suppression of the function of a single hemisphere of the cerebellum has no constant recognizable symptoms (*Cbl.*, 1876, p. 387); and therefore local lesions affecting only one-half cannot be diagnosticated. It is probable that these structures have some connection with psychic processes, but we need further observations before we can say certainly in regard to this point,

In many cases of cerebellar disease we meet with disorders of co-ordination, but this is only the case when it affects directly or indirectly, the vermis, the middle lobe of the cerebellum. As soon as disease of the cerebellum, or, especially, morbid processes developing in the posterior fossa of the cranium, begin to exercise pressure on the vermis, will the symptoms of cerebellar ataxia make their appearance, and they will be lacking when this is not the case. Why, in these cases, the upper extremities usually are uninvolved, is not yet clear.

We await a fuller exposition of the above mentioned points, and an exhaustive description of the cerebellar disorders of co-ordination, with the points of distinction between these and similar spinal symptoms, in a forthcoming memoir by the author.

DIPSOMANIA.—At the annual meeting of the Border Counties Branch of the British Medical Association, July 26th, 1878 (reported in *Brit. Med. Journal*, Nov. 16), the President, Dr. Gilchrist, read a paper on the following subject: Dipsomania: its Nature, Treatment, and Results. The conclusions arrived at were the following: 1. Dipsomania is a true neurotic disease. 2. In a very large proportion of cases, if not in all, it is hereditary. 3. In a large number of cases, it is traceable to drinking habits in one or both parents, or connected with them in more remote relationships. 4. Its neurotic character is indicated—*a*, By its tendency to periodicity ; *b*, By its incurability; *c*, By the loss of self-control ever a dominant propensity in those subject to it; *d*, By the intellectual, and especially moral, peculiarities of its victims. In the above characteristics are seen its intimate relationship to, if not identity with, ordinary insanity. 5. In its treatment there are to be recognized two essentially distinct conditions: the neurotic peculiarity, which is permanent and irremediable, and the direct results from the poisoning of alcohol, which are temporary and remediable. 6. If the term "recovery" is to apply to the latter conditions only, 90 to 100 per cent. are curable. If it is to include the former also, 5 per cent. will be found a liberal allowance; that is, not more than five per cent. of the patients will be so far modified by any treatment as to enable them to resist, during the remainder of life, the propensity to drinking. Dipsomania, like other forms of insanity (neurotic diseases, if preferred), is a sad inheritance from the sins of our fathers. It is very rarely the result *merely* of vicious habits in the individual subject to it. If it is to be dealt with effectually, we must extend the horizon of our views beyond individual cases, or even the present generation. Prevention *versus* cure is the true, if not the only treatment.

DISLOCATION OF THE ATLAS.—Drs. Uhde, Hagemann and Boettger describe in the *Archiv f. klin. Chirurgie*, vol. XXII., a case of bilateral wrench of the lower articular surfaces of the atlas, by which the right surface was displaced forward, the left backward, from corresponding articular surfaces of the axis. The injury produced some remarkable disturbances of innervation. The left half of the tongue was paralyzed and convex outwards, while the right half was contracted and concave. The left half of the soft palate and the left glosso-palatine arch were also paralyzed, the uvula was drawn to the right. The anterior third of the tongue on both sides and the second and third thirds on the left, possessed ordinary and gustatory sensation ; in the posterior two-thirds on the right, no sign of sensation or taste could be detected. The authors attribute these phenomena to paralysis of the right glosso-pharyngeal nerve and of the left hypoglossal nerve and the pharyngeal plexus. By artificially producing the injury, they show that the glosso-pharyngeal nerve, immediately after its exit from the jugular foramen, must have been violently stretched over the portion of the atlas which was thrown forward; that in this luxation, the roots of the hypoglossal nerve inside the vertebral canal appeared as a pair of tightly stretched cords, instead of lying, as in the normal condition, loose against the dura mater. In the same way and at the same part, the accessory nerve suffers stretching, leading to the paralysis of the left palate; this can be explained in no other way than by assuming an injury, through this overstretching, of the pharyngeal plexus, which aids in forming the anterior branch of the accessory nerve. The vertebral canal was not so much narrowed as to produce compression of the medulla oblongata, and the vertebral artery was not injured. The disturbances of the gustatory function of the tongue indicate that the glosso-pharyngeal nerve supplies exclusively only the two posterior thirds of the tongue, and that the anterior third and palate are supplied from portions of the third division of the fifth nerve. The dislocation could not be reduced; but the patient gradually regained some power of moving the head. There was also some improvement in the functions of the tongue and palate.—*Brit. Med. Journal*, Sept. 7, 1878.

HALLUCINATIONS.—Stricker, *Wiener med. Blaetter*, 1878, No. 4-6 (abstr. in *Centralbl. f. d. Med. Wissensch.*).—As regards the question of the production of hallucinations, the author thinks we can only consider whether the nervous apparatus for the conceptions can act alone, or whether the peripheral nervous apparatus is not also requisite. Adult inmates of an institution for the blind state that the conceptions of colors and forms occur in dreams for a period of ten to twenty years after the onset of total blindness; then auditory and tactual conceptions gradually predominate, until finally all sight is lost in dreams, though the recollections of objects seen in childhood still persist. This indicates that that part of the central sensory organs that serves in the memory of sensations is not alone sufficient to produce hallucinations. Something else must intervene in their production. He compares the hallucinations of sound individuals in the state of semi-conscious condition, when the functions of the brain are in a condition of lessened irritability and those of insane persons which also accompany a depressed condition of consciousness. The peripheral nerves, he thinks, are needful for their production ; in actual perceptions their centres are put into action through their agency by external irritations, but in hallucinations it is an internal excitation from the centres that excites the peripheral sense apparatus.

VERTIGO DUE TO URETHRAL STRICTURE.—In Nos. 44 and 45 of the *Deutsche med. Wochenschrift* (1878), Dr. Erlenmeyer relates a case of vertigo which could be traced to the above mentioned cause.

A healthy man of 31 years, without any hereditary nervous tendency, began to complain about six years ago, of sudden attacks of stiffness of the left arm. They were of momentary duration, but soon became more frequent and involved also the left leg and right arm. After a time the attacks were accompanied by sudden vertigo, without loss of consciousness. A feeling of pressure in the forehead now became a constant complaint. The patient suffered from urethral stricture, rendering micturition quite difficult and every attempt at micturition was accompanied by dizziness, without motor troubles. Examination showed catarrh of stomach and bladder, and increased patellar reflex. On closing the eyes he walked unsteady and complained of vertigo; but no other symptoms of ataxia existed. Treatment with nitrate of silver removed the gastric catarrh, but had no influence on the other troubles. These, however, were *definitely removed by dilatation of the stricture.* A final examination revealed only an increased patellar reflex, slight pains in back and early fatigue on walking. Closure of the eyes still induced unsteady gait and vertigo, which, however, never occured under other circumstances.

THE PATHOGENY OF GENERAL PARALYSIS.—The following are the concluding paragraphs of a communication by M. Luys on the pathogenic conditions of the development of general paralysis and of the sclerotic degenerations of the nervous system, read before the Societe Médicale des Hopitaux, April 12, 1878, and published in *L'Union Médicale*, Oct. 1 and 8, 1878 :

En resumé, the interstitial frame-work which serves as the support and trophic agent to the elements of the nervous system, is, in the living person, from birth to old age, in a constant evolution, which manifests itself appreciably by a hardening and progressive condensation of the fibres.

This continual work of production of new elements appears to be at its maximum intensity between the ages of thirty and fifty years. After the latter period it remains stationary.

1. The elementary lesions constituting general paralysis are only a morbid exaggeration of this normal process. The maximum frequency of this disease, as regards its time of appearance, corresponds with the epoch when the process of development of the neuroglia is most intense.

The influence of external causes acts in the production of reiterated hyperæmias and consequent irritation of the corpuscles of the neuroglia. They whip up, so to speak, the natural process of evolution, which thus becomes their accomplice.

2. Other disorders of the nervous system are due to the same organogenic conditions. It is always the case that the neuroglia, in its various forms, takes part, more or less, in the degenerative process ; as is seen in the sclerotic lesions of locomotor ataxy, paralysis agitans, multiple sclerosis, etc.

In a more general point of view the process of sclerotic hyperplasia that is developed in the intimate structure of the nervous system, is only the diminished expression of a series of other similar processes that occur elsewhere in the different viscera of the organism, under the form of sclerotic degeneration. The general laws of morbid evolution manifest themselves everywhere in the same manner ; there is always the same insubordination of the passive element of the interstitial fibrous frame-work, living in the closest relations with the special elements of the tissues that crowds out these latter and substitutes itself in its place.

SYPHILITIC EPILEPSY.—The following are the conclusions as to the diagnosis of the syphilitic origin of epilepsy, derived from an analysis of two hundred and seventy-four cases, by Dr. Thos. Stretch Dowse, *Practitioner*, Oct., 1878 :

"The age of the patient is an important guide. Should a man or woman be attacked by epilepsy between thirty and forty years of age, without having any hereditary predisposition, or a previous seizure, then a syphilitic cause may be apprehended. And, apart from this, provided that between the attacks there is more or less mental derangement, our basis for a diagnosis is greatly simplified, and it is even more so if there be a paresis more or less profound, localized or unilateral, but gradually passing off after the epileptiform seizure. The reflex processes are rarely, if ever, completely absent. The iris may contract under the influence of a strong light; the lips close when the conjunctiva is tickled, and a state of sub-consciousness rather than profound coma is a prominent feature from first to last.

"The stages of the attack are ill-defined, and merge the one into the other. The universal tonic spasm, with thotonism rarely presents itself. Pallor rather than cyanosis is the facial exponent, and the duration of the fit is protracted sometimes to many hours, with intervals of wandering, delirium and excitement. Foaming at the mouth is less common in these cases than a profuse flow of saliva, and all sorts of cries are associated with the seizure; but they are rarely so exalted as Romberg expresses it, 'shrill and terrifying to man and beast.'

"And lastly in reference to albumen in the urine. I have given considerable attention to this point, and I have failed to find it present in any but a few of the cases; but epileptoid seizures, associated with albuminoid syphilis and a plentiful secretion of phosphatic albuminous urine, are not uncommon."

HAY FEVER.—Dr. Geo. M. Beard, *N. Y. Med. Record*, Nov. 2, insists on the neurotic character of hay fever, and the non-essential character of particular symptoms. He says, "Just as in writer's cramp—a disease I am now specially investigating—there are many cases that do not have the characteristic cramp; just as there may be sick headache without the usual nausea and vomiting, just as we may have locomotor ataxy without the ataxic gait, just as there may be hysteria without hysterical convulsions, just so there may be genuine hay fever without any of the familiar symptoms of the nasal respiratory tract.

He states that among other anomalous cases of the disease, he has met with those in which the usual symptoms were replaced by conjunctivitis, eczema, spasm of the œsophagus, and in one a transient melancholia preceding by a few days the ordinary attack, during which the patient was insensible to the effect of large quantities of alcohol, though at other times easily affected by it.

SCLEROSIS OF THE SPINAL CORD AND THE RESULTS OF MYELITIS.—E. Leyden gives, in the *Charité Annalen*, for 1878, the results of some experiments on the artificial production of sclerosis of the spinal cord with the view of determining, as far as possible, whether it is to be regarded as a special form of chronic myelitis, and what are its relations to acute myelitis. By injecting a few drops of Fowler's solution into the spinal cord of dogs, he was able to excite violent inflammation. One of the animals operated on survived fourteen months; notwithstanding total paralysis and contracture of the hind limbs, its general health was good. After the animal was killed, a careful examination was made. The spinal cord at the seat of operation was very thin, and was covered with a thick layer of fat; in the substance of the anterior cornua was a cyst, having its origin in the softening of the tissue; there was also found a focus of rarefied matter, consisting of loose connective tissue with dead nerve elements; and in the neighborhood of the focus of softening, in the parts which, at the beginning of the process, were swollen and infiltrated with cells, was a tough sclerotic net-work containing only scattered nerve fibres, without nucleated cells, but with some stellate cells and single nuclei. The muscles of the hind leg were the seat of interstitial fatty growths; the muscular fasciculi had almost disappeared; the motor nerve roots were atrophied; the degeneration extended upward in the form of a narrow strip in the posterior columns as far as the cervical enlargement. The sclerosis arising from acute myelitic processes or produced by experiment on animals, corresponds to the sclerosis observed in the human spinal cord, and appears to be an ultimate product of those inflammations of the spinal cord, which, without essential distinction, lead to infiltration, and subsequently to atrophy.—*Brit. Med. Jour.*, Nov. 9, 1878.

SYPHILITIC NEUROSES.—We copy the following abstract of a paper, by Dr. W. R. Gowers, read before the British Medical Association last August, for the *British Med. Journal*, Aug. 31:

The object of the paper was to examine the question of the extent to which syphilis is a cause of the affections of the nervous system, distinguished as neuroses, from the diseases of the adneural structure, membranes, vessels, etc., which are known to be so frequently syphilitic. The conclusions were drawn from personal observation at the National Hospital for the Paralyzed and Epileptic, all cases being rejected in which any indication of primary "adneural" disease could be traced or in which any other cause than syphilis could be assigned. The conclusions reached were that idiopathic epilepsy does not result from acquired syphilis. Almost all cases of chronic convulsion after syphilis presenting indications of primary local brain disease, or else of inherited tendency to epilepsy. On the other hand, idiopathic epilepsy was found to be an occasional result of inherited syphilis. No case of the association of chorea and syphilis, inherited or acquired, had been met with, although a few cases are on record in medical literature. Certain forms of general paralysis certainly followed syphilis, but they rarely presented marked optimism. Paralysis agitans cannot be associated with syphilis, but progressive muscular atrophy seems to be an occasional distinct effect, although in inferring this, care must be taken to avoid mistaking for it the results of pachymeningitis. Lateral sclerosis of the cord, it was thought, only occurs as a consequence to syphilis secondarily to pressure higher up, but posterior sclerosis (locomotor ataxy) is certainly one of the most common neural effects, many syphilitic cases having been met with in which there was no evidence of pressure or initial myelitis, but there had apparently been a primary slow degeneration of the posterior columns. Cases illustrating the several affections were appended to the paper. Dr. Gowers had found that most of the cases considered in the paper were not amenable to anti-syphilitic treatment in the same degree as the adneural diseases ; but in most of the cases the diseases had existed for some time before they came under observation, and the importance of the trial of early treatment by those who had the opportunity was strongly urged. If it were found that in these affections, as in some of the late manifestations of inherited syphilis, tonic rather than specific treatment was needed, it was probable that the connection with syphilis was an indirect one. Some observations on the prognosis in the common syphilitic diseases of the nervous system (of membranes, vessels, and growths) concluded the paper. It was urged that more caution in prognosis is necessary, by discrimination of the adneural syphilitic disease, which could be cured, from the neural effect, which was not syphilitic and was often beyond treatment.

THE MENSTRUAL PSYCHOSES.—The following is the abstract of a paper by Dr. Krafft-Ebing in the *Archiv f. Psychiatrie*, VIII., 1, 65, as given in the *Revue des Sci. Méd.*, Oct., 1878 :

The author gives his first pages to the collection of certain facts scattered through medical literature, and relating to psychoses occurring periodically at the menstrual periods. He adds to these nineteen observations of his own, of this kind of insanity, in which the symptomatology, the march, the prognosis, and the effects of treatment (in particular, the administration of bromide of potassium) are given in full detail.

He next discusses the pathology of menstrual psychoses. All agree in admitting that in females, at the period of menstruation, the nervous system exhibits a certain greater vulnerability than is its habit ; hence it might be questioned whether the psychic disorders coming on at these periods, are any more than an exaggeration of the phenomena of excitation usually observed in females at this period of their physiological existence. But facts of clinical observation show the intervention, in menstrual psychoses, of a predisposition of older date which renders the intellectual centres a veritable *locus minoris resistentiæ.* In eleven of the cases observed by Krafft-Ebing, the influence of heredity was very manifest, and in all those in regard to whose antecedents particulars could be gained, were exhibited all the signs of a neuropathic constitution, with disorders of sensibility, of motility, and of the vaso-motor system.

On the other hand, as the functional disorders of menstruation and the lesions of the genital organs are not always very marked, and may even be lacking, it is evident that the physiological process of menstruation is, of itself, capable of provoking an acute psychosis in a woman whose brain is in a condition of abnormal excitability.

As to the pathogenic mechanism of these psychoses, they are evidently vaso-motor neuroses, characterized by periodical congestions of the cerebral centres. It remains to be seen how the menstrual process can develop phenomena of fluxion in the brain, analogous to those that have the genital organs for their locality. But the menstrual fluxion, according to Pflueger, is only a reflex process, having its point of departure in the irritation of the ovarian nerve, which is transmitted next to the central organs of the nervous system. It is therefore admissible that, under pathological conditions, this ovarian nerve excitation irradiates over a greater extent of the nervous centres, and that it involves vascular territories, that under normal conditions would not have been implicated.

Menstrual psychoses may develop themselves at any epoch of sexual life.

Considered in point of view of their symptomatology, the menstrual psychoses are grouped in two principal clinical classes: the melancholic and the maniacal, the latter being the most frequent. There is nothing specific in their symptoms. It is only in an etiological point of view that they require any distinct place in mental pathology.

In the interval of the paroxysms we observe a prostration with physical and mental exhaustion, the duration of which is in relation with the intensity and duration of the paroxysms themselves, and in addition the symptoms of a neuropathic constitution.

As regards their progress, the paroxysms have a tendency to return at each menstrual period. But it is not uncommon for them to be suppressed for months and even years, when the patients' constitutions are improved and they are placed in good hygienic conditions. Nevertheless the prognosis, like that of periodical psychoses generally, is not very favorable.

The first point in the treatment is to fulfill the causal indications. We must treat the neuropathic constitution of the patients and seek to diminish the impressionability of the brain centres, by improving the nutrition, protecting the patients from excitement and especially genital excitations, and treating uterine disorders if there are any.

Krafft-Ebing has made a trial of bromide of potash in the treatment of these affections, employed as a preventive, between the paroxysms; the medicine has given lasting success six times in nine cases; in three others the success has been only temporary. Even when given during the attack the bromide has given good results five times out of nine, in the other four the results were negative. For the medicine to act the daily quantity should not be less than six grammes. Opium, morphine, and chloral are shown to be absolutely inefficacious in the treatment of menstrual psychoses.

THE PATHOGENESIS OF CEREBRAL HEMORRHAGE.—Eichler, *Deutsch. Arch. f. klin. Med.*, XXI., 1–32. (Abstr. in *St. Petersburg. Med. Wochenschr.*, No. 42, 1878.)

The author first gives an extended historical *resumé* of the changes in the views regarding cerebral hemorrhage, and thoroughly examines the view of Charcot and Burchard that miliary aneurisms of the smaller arteries supply the cause of the hemorrhage. On the incitation of Prof. Heller. of Kiel, he examined 3–400 miliary aneurisms, and among others those taken from two brains by himself. These miliary aneurisms are due to an arteritis, affecting especially the intima. The following are his general conclusions :

1. Primary idiopathic cerebral hemorrhage owes its production to the rupture of miliary aneurisms of the smallest cerebral arteries.

2. The miliary aneurisms are *aneurismata spontanea vera totalia.*

3. They are due to a chronic endarteritis identical with arterio-sclerosis.

4. Miliary aneurisms, like the arterial sclerosis are pre-eminently senile diseased conditions.

5. The dissecting aneurisms are sharply separated from the miliary aneurisms. They are simple hæmatoma, and not a cause, but a consequence of hemorrhage.

6. The capillary dilatations (capillary aneurisms of Schroeder Van der Kolk) are likewise distinct from the miliary aneurisms. They are to be compared with telangiectases of other organs, and like them are congenital.

7. The coats of the vessels are in three layers: the intima, the media, and an externa, separated from the muscularis by a lymph space.

A MODERN MIRACLE.—Seldom have miracles been performed under such favorable test conditions, and subject to such unbiased record as the following, which is reported by Surgeon A. Crombie in the *Indian Medical Gazette:*

"A case of cholera was admitted to the Mitford Hospital in April, 1878. The man was dumb, and could give no account of himself. His name, age, caste, occupation were unknown, and these headings remained unfilled up in his bed ticket till he was discharged. The case was one of average severity, with the usual amount of thirst, cramps and general distress, but during the whole time he was under treatment he did not utter a single articular complaint. He endeavored to engage the attention of the ward-coolies from time to time, by signs, to give him water, and to rub his cramped limbs. He was treated in the ordinary manner with hypodermic injections of chloral, to relieve the spasms, with stimulants, and finally with full doses of calomel 'to restore the biliary function.' Relays of students sat at his bedside to administer his medicine, take his temperature, and give the subcutaneous injections, and Mr. McNaught, the house surgeon, saw him three or four times every day. He was at last discharged cured after fifteen days' stay in hospital. Not a doubt remained on the minds of those brought into contact with him, and who had sat at his bedside for hours, at the time witnessing the severe ordeal of cholera agony through which he was passing, that the man was dumb, and but for subsequent events his would have passed into the great limbo of recorded and forgotten cholera cases.

"It would be hard to conceive conditions better adapted to be the preliminaries of an indisputable exhibition of miraculous power than these. The man was involuntarily subjected to the keenest bodily agony for days together, during which he was removed from possible accomplices, and under the constant observation of educated and critically disposed persons. If there had been mendacity in the case, it must have betrayed itself.

"It may be judged, then, with what surprise this dumb man, ten days after his discharge, was found to present himself in the out-patient department, talking freely and bringing with him the aged devotee who had restored his speech, but who it seems was a martyr to dyspepsia. There was no reticence with either of them as to the visible means by which the miracle had been performed. The ancient Brahmin had taken a handful of the ashes of the fire with which he had just cooked his meal of rice, and had rubbed them on the dumb man's breast, and that instant he found he could speak, after having been dumb for three years. He did not now bring him to the hospital because of the wonder he had accomplished, but that the old man might get the benefit of European treatment for the cure of his dyspepsia. The incongruity of the conjunction of these two ideas did not seem to have occurred to them till it was chaffingly pointed out to them by the bystanders.

"The case, it seems, had been one of demoniacal possession. Eight years ago this man had gone one evening to a tank to catch fish for his evening meal. The tutelary demon of the place and of the fishes had, however, thrown him down and entered into him, and from that time till he made our acquaintance at the Mitford Hospital he had been sometimes dumb, sometimes deaf, sometimes blind, and often convulsed. During the whole of the time, he had suffered also from persistent otorrhœa, which had been cured at the same time that his speech was restored. He had been continuously dumb for three years at the time he was seized with cholera and sought admission to the Mitford Hospital.

"In demonology, nothing is so striking as the extreme simplicity of the means by which the most wicked of evil influences may be frustrated and the demons themselves exorcised. Homœopathy contains nothing approaching it. A line drawn on the ground with a stick, a horse's shoe nailed on a door, are effectual bars to the most malicious fiends, and a pinch of salt over the left shoulder suffices to thwart the most devilish designs. But in this man's statement regarding his otorrhœa, of which there is unfortunately no corroboration in the clinical record of the case, may we not (asks Dr. Crombie) be pardoned in fancying we see the least glimmering of light on the possible *rationale* of this seeming miracle?"—*British Med. Jour.*, Oct. 12, 1878.

THE following are some of the titles of recently published papers on the Pathology of the Nervous System and Mind and Pathological Anatomy:

LYKE, Dipsomania, *Hospitals Tidende*, Nov. and Dec., 1874.—MARANDOR DE MONTYEL, The Progress of Progressive General Paralysis in Hereditary Cases, *Ann. Med. Psych.*, Nov., 1878.—HIPPOLYTE MARTIN, The Mortality of the Children of Epileptics, *Ibid.*—CHARCOT, A Lecture on Certain Phenomena of Hysteria Major, *Brit. Med. Jour.*, Nov. 30.—GUITERAS, Lectures on a case of Facial Monoplegia, Illustrating the Localization of Cerebral Functions and Lesions, *Philad. Med. Times*, Oct. 26 and Nov. 9.—HAMILTON, The Value of Absent "Tendon Reflex" as a Diagnostic Sign in Locomotor Ataxia, with an Analysis of Eight Cases, *Boston Med. & Surg. Jour.*, Dec. 19.—FIELD, Dermalgia from Quinine, *N. Y. Med. Rec.*, Nov. 30.—CONNOR, Hystero-Epilepsy, *Detroit Lancet*, Dec.—FULLER, Exophthalmic Goitre, *Ibid.*—LUNDY, Optic Neuritis, *Ibid.*—MACSWINEY, A Fatal Case of Railway Spine, *Dublin Jour. Med. Sci.*, Dec.—BEARD, Are Inebriates Automatons? *Quarterly Jour. of Inebriety*, Dec.—EASLEY, A Study of

Shock, *Richm. and Louisv. Med. Jour.*, Nov. and Dec.—BUZZARD, Clinical Lectures on Some Points in the Diagnosis of Spinal Sclerosis, *London Lancet* (Am. Repr.), Oct. and Nov., 1878.—MYERS, The Influence of the Nervous System on the Health of the Mouth, *Lancet and Clinic*, Dec. 21.—BEARD, The Scientific Lessons of the Mollie Fancher Case, *N. Y. Med. Rec.*, Nov. 7.

c.—THERAPEUTICS OF THE NERVOUS SYSTEM AND MIND.

GLEDITSCHIN.—B. F. Lautenbach, *Philadelphia Med. Times*, Nov. 23, gives an account of the results of a series of investigations on the fruit of two species of Gleditschia growing in the parks of Geneva, Switzerland, where he is now engaged as assistant to Prof. Schiff.

In these investigations it was found that a watery, alcoholic, or ethereal extract of the ripe fruit and seeds of these trees exerted almost no toxic influence on frogs and toads. When, however, an alcoholic extract of the unripe seeds and the portions of the fruit immediately surrounding these was used, very active poisonous effects were observed. In from five to twenty minutes the frogs were in a profound state of stupor. No reflex movements could be excited by any of the known means, though at that time the motor nerves still remained irritable. This loss of reflex activity was not due to loss of function of the sensory nerves through the direct action of the poison on these structures, as, after ligature of all the blood-vessels of a limb, irritation of that limb failed to produce reflex movements when the animal was poisoned with the extract. The heart continued to beat for hours after these symptoms appeared. If a not too large dose was given, the animals recovered after having been in this state for twenty-four hours.

The question now was to determine the nature of, and isolate the principle, on which the activity of this extract of gleditschia depends. Were it a glucoside, it would almost certainly be extracted by water. Watery extracts, hot or cold, are, however, inert if no alcohol was used in their preparation. Again a solution of the extract from which the coloring matters, the tannic acid, and the inorganic salts have been removed, does not, as do all glucosides, yield glucose when boiled with dilute acid.

Further investigation showed the active substance to be an alkaloid. Gleditschin, as he proposes to call this new alkaloid, forms salts with sulphuric, nitric, hydrochloric, acetic, and tannic acids. All of their salts crystallize in modifications of the rhomb.

The facts determined thus far respecting the physiological action of this substance are hereby briefly given ; in a later publication this subject will be treated more fully.

Locally applied to the blood of mammals, like saponin, it causes the blood-corpuscles to disappear. These corpuscles, however, later reappear.

The hearts of frogs continue to beat long after all other signs of like have disappeared. The vagi of frogs poisoned with gleditschin still retain their inhibitory action on the heart. The first symptom produced in frogs is a state analogous to sleep. Following this, rapid abolition of reflex activity takes place, and respiration ceases. The galvanic irritability of the nerves is much diminished.

ERGOT IN SUNSTROKE.—Dr. R. F. Dedrickson reports, *Dublin Jour. of Med. Sci.*, Oct., 1878, two cases of heat apoplexy treated by him last July in Calcutta, with ice at the nucha, fluid extract of ergot internally. In one case, where the cardiac action was strong, he added three minims of tincture of aconite to the fifteen minims of ergot given every hour. A good recovery was obtained in both cases. Several less severe cases were similarly treated with success.

Dr. Dedrickson suggests that in cases where the power of swallowing is gone, the hypodermic injection of ergotine should be tried.

PILOCARPIN IN ECLAMPSIA AND AS AN OXYTOCIC.—*Centralblatt fuer Gynäkologie*, Nov. 15. Dr. E. Bidder, St. Petersburg, records two cases of eclampsia in which pilocarpin was repeatedly used, giving little relief or any to the convulsions, and producing no uterine contraction. Dr. E. Wellponet, St. Petersburg, also records a case in which the most careful and repeated employment of pilocarpin in large doses, failed to induce labor, though it produced the physiological effects of jaborandi. (*Edinburgh Medical Journal*, Sept., 1878.)

THE THERAPEUTICAL ACTION OF GELSEMIUM.—Professor Massini, of Basle, has published a small pamphlet giving an account of eighty cases of neuralgia of the trigeminus, treated by full doses of the tincture of gelsemium. He prefaces his remarks with a brief description of the physiological action of the drug. Redness of the conjunctiva, pain in the eyelids, contraction of the pupil, double vision and giddiness, are the symptoms which generally follow the administration of moderate doses. When the dose is increased, slight ptosis, dilatation of the pupil, gaping, languor, and pain in the limbs are the usual results. The respiration is not affected. In frogs, on the other hand, a large dose produces paralysis of the respiratory muscles, the heart's action remaining unchanged. In cases of neuralgia of the trigeminus, Dr. Massini gives twenty minims of the tincture every half hour up to three doses, and he finds that the first dose usually affords relief, and that the pain rapidly subsides after a second or third dose has been taken. He has never found it necessary to exceed sixty minims, and only in one case did this quantity produce unpleasant head symptoms. The cases in which the remedy produces most benefit are those of simple rheumatic neuralgia of the alveolar branches of the trigeminus; in those it rarely fails. It also sometimes relieves the pain remaining after the stopping of a carious tooth. Where there is any inflammatory affection of the bone or periosteum no good can be expected from the remedy. The medicine may, if necessary, be repeated several days in succession, the active principle rapidly passing off by the kidneys. (*Medical Examiner*, August, 1878.) (*Edinburgh Medical Journal*, Sept., 1878.)

THE ANTAGONISM BETWEEN ALCOHOL AND STRYCHNIA.—Dr. Stacchini, from observing some cases where the administration of alcohol seemed to be of service in arresting the action of strychnia, was led to investigate the question whether there was any real antagonism between them: he experimented on frogs, Guinea-pigs, and dogs. His results were essentially negative; he found that alcohol cannot be regarded as antagonistic to strychnia. Alcohol, nevertheless, produces diminution in the violence of the convulsion, and retards the access of death under circumstances that would otherwise prove fatal. Animals die after the lapse of a longer or shorter period, notwithstanding the absorption of a large amount of alcohol, if the doses of strychnia have been sufficiently considerable. Alcohol is, perhaps, the least dangerous of the remedies that are used in strychnia-poisoning, its own action, either injected in a diluted form, or injected subcutaneously, is certainly less dangerous than that of chloral introduced in the same way. A fatal dose is not interfered with by strychnia, nor does strychnia seem to modify the inebriating effects of alcohol. If alcohol and strychnia are given together in an absolutely fatal dose, the animal dies, presenting the combined symptoms of both. (*Gazetta Med. Ital. Imparziale*, Nov. 9, 1878.) (*Practitioner*, Nov., 1878.)

TREATMENT OF NEURALGIA BY HYPODERMIC INJECTIONS OF ERGOT.—Marino recommends the injection of from 0.15 to 0.25 gr. dissolved in six grains of distilled water. This may be repeated once or twice, though perhaps not more than six times, and acted well in certain forms of neuralgic pains, especially in tic douloureux. It appears to act less favorably in sciatica. (*Imparziale*, No. 8, 1878.) (*Practitioner*, Nov., 1878.)

CEREBRAL ELECTRIZATION.—We find in the *Gaz. des Hopitaux*, 1878, No. 119, an abstract of a paper read before the French Association for the Advancement of Sciences, last year by M. Letourneau, giving the results of his clinical experience in electrization of the brain in various mental disorders. He first undertook a series of experiments on animals, on the vaso-motor nerves of the head. The most noteworthy result was the ascertainment of the fact that electrization of the superior cervical ganglion produced constriction of the retinal vessels. This was already observed in man, and M. Letourneau had himself noticed it in a case he was treating for choroiditis. Deeming that the condition of the retinal circulation was an index of that of the brain, he concluded that the vertigo produced by passing an electric current through the head was due to narrowing of the cerebral vessels and consequent cerebral anæmia, and not congestion as had been thought to be the case by some. He therefore applied this deduction to practice and treated by electrization of the head a very intelligent patient suffering from a tendency to cerebral congestion, and with very good success. Each electrization, whether of the head directly or of the superior cervical ganglion, caused immediate relief. The patient was relieved of his "cap of lead," as he expressed, his sight became better and objects appeared more brightly colored. Other cases were similarly benefited.

A marked tendency to sleep was often observed to follow the application. He also found it beneficial in dissipating melancholia in two cases.

He concludes from these facts that cephalic electrization is indicated in a vast number of mental disorders, especially when of recent date. It would seem to be *a priori* indicated in some cases of acute mania.

We should hesitate to recommend this treatment, except with the utmost caution to avoid too strong currents and watch carefully for both good and bad effects.

NERVE-STRETCHING IN LEPROSY.—Dr. Edward Lawrie has stretched the ulnar nerve in thirty cases of anæsthetic leprosy. In every instance the operation was followed by benefit so far as the area supplied by the nerve was concerned. (*Indian Med. Gazette*, Sept., 1878.) (*Phil. Med. Times.*)

THE ARSENICAL TREATMENT OF CHOREA.—Dr. L. C. Gray, *Proc. Med. Soc. Co. of Kings*, Dec., 1878, discusses the treatment of some twenty-seven cases in his dispensary practice with arsenic. Six of these he was able to keep continuously under his observation from the beginning of treatment till the entire disappearance of all the symptons. He used in all cases Fowler's solution, and as a vehicle Huxham's tincture of cinchona. The doses of the Fowler's solution were three drops *ter die* after meals in the beginning, gradually increasing one drop every second day until there was distinct abatement of the disease or slight toxic symptoms. No directions whatever were given as to hygiene or food.

The average duration of the six cases from the beginning of treatment to complete recovery was twenty-four days, the maximum thirty-eight days, the minimum thirteen days, and the average time from the beginning of symptoms (as stated by friends) to recovery was fifty-five days, the maximum one hundred and three days, the minimum twenty-one days.

Comparing these figures with the results of the expectant treatment, lately so much lauded by Drs. Gray and Tuckwell, we have a difference in favor of the arsenical treatment of twelve days in the duration of treatment over those reported by Gray and Tuckwell, and by Wilks as treated by the expectant method, even including in these latter, as was not done in the others, the duration of the disease before coming under treatment. As a corroborative evidence of the value of the arsenical treatment, and as agreeing with his own figures, he quotes the fact that the average duration of twenty cases of chorea thus treated at St. Thomas' Hospital, London, was 26 days.

The credit of the first suggestion of this treatment, belongs, according to Dr. Gray, to Dr. D. M. Reese, of Albany, N. Y., who recommended it strongly in 1839, and not as is commonly supposed, to Dr. James Begbie, of Edinburg, whose paper was read in 1858.

A comparison of the arsenical with other forms of treatment led the author to the following conclusions : First he places arsenic, then sulphate of zinc, and next iron, and he thinks the best results would be obtained by a judicious combination of two or more of these remedies, with good hygiene and a sufficiency of nourishing food. In dispensary practice, of course, this last cannot always be relied on, and then the relative value of drugs is a matter of importance.

BROMIDE OF POTASSIUM AS A DIURETIC.—In the case of a gendarme suffering from albuminuria and uræmic convulsions, Dr. Sohier administered bromide of potassium in doses of thirty to forty-five grains. Free diuresis was produced, between two and three quarts of urine being passed in one night, and the albumen, which was previously present in large quantities, disappeared entirely. In three other cases the bromide produced similar diuretic effects. Its diuretic action is exerted on healthy persons also. (*Centralbl. f. Chirurgie.*) (*N. Y. Med. Record.*)

TREATMENT OF HYSTERICAL REFLEX NEUROSES.—Prof. Weber recommends the protracted employment of chloroform inhalations in the treatment of obstinate and severe cases of hysterical reflex neuroses of the respiratory apparatus when the primary seat of irritation cannot be discovered and treated. He has himself proved the value of the inhalation in several cases. His first case was of a lady with a spasmodic cough that had proved rebellious to all treatment ; she was cured in eight days by the chloroform inhalations, which were administered as often as the cough came on. A child with sneezing spasms was cured in three days by the chloroform. Another lady with a spasmodic cough was treated with the same remedy for fourteen days, the inhalations being administered at first four or five times, and afterwards two or three times daily. She was much improved ; the cough only came on after a walk, and the inhalations were only required then. In four weeks she was discharged cured ; a subsequent slight relapse was cut short by the internal administration of chloroform. In the case of a girl, fourteen years of age, who suffered from spasms of sneezing, the inhalations produced a speedy cure.—*Memorabilién* (*N. Y. Med. Record*).

QUININE DERMALGIA.—Prof. H. M. Field, of Dartmouth, in the *N. Y. Med. Record,* Nov. 30, gives the account of a case in which large anti-periodic doses of quinine produced in the cutaneous surface great heat, local œdema, especially in the face and hands, hyperæsthesia, followed in a few days by desquamation. With these symptoms were associated more or less gastric disturbance, and on one occasion, violent vomiting, headache, and delirium. The patient preferred the original disease to that produced by the remedy.

The course followed by Dr. Field was to give the medicine in greatly lessened doses, which had the full therapeutic effect, with only a light reminder of their former unpleasant effects.

From such facts as this Dr. Field thinks we have an indication as to the physiological action of the drug. We are not, of course, to believe that it is excreted by the skin, there is no sufficient evidence pointing that way. But he holds that it does act chiefly on the sympathetic and not on the cerebro-spinal systems, and that these facts of dermalgia, urticaria, œdema, etc., are proofs that such is the case.

Its particular influence on the skin, as noted by Erasmus Wilson, and others, he deems has been thus far insufficiently studied. He is convinced that certain remedial resources of the agent in this direction still await investigation.

CANNABIS INDICA IN EPILEPSY.—Dr. Wharton Sinkler, *Phil. Med. Times*, Sept. 28, reports having used cannabis indica in two cases with advantage, one of which is given in detail: A boy, ten years of age, suffered from epilepsy which commenced as *petit mal*, occurring once or twice a day for two months, then changing to (chiefly) nocturnal *grand mal*, occurring about every two hours. He was treated with potassium bromide, and tincture of the chloride of iron, which, however, only modified his attacks, but gave no permanent relief; as soon as the medicine was stopped the attacks were more severe than ever. Then oxide of zinc was tried, but no good effect was produced. This was discontinued and cannabis indica, one-sixth grain three times a day, was ordered, with the result of first decreasing the number of his attacks to one a day for a week, and then stopping them altogether. At the date of the report there had been none for nearly three months. The child's intelligence and disposition, which had before been much injured by this disease, were also wonderfully improved.

A second case was also improved, but the full effects of the drug were not yet tested upon it.

CONIIN.—The following is the abstract of a thesis by M. Tiryakian, *Thése de Paris*, 1878, as given in the *Revue des Sci. Méd.*, Oct.:

Coniin, conicine, or cicutine is the active principle of conium maculatum (hemlock). M. Tiryakian has undertaken the study of this alkaloid, with the collaboration of M. Bochefontaine, and under the direction of M. Vulpian. The experimental results obtained differ considerably from those of MM. Martin-Damourette and Pelvet, Christison and Gubler; the author attributes this difference to the mixture of the commercial coniin with foreign substances; it contains especially an essential oil, as yet little known, that produces in poisoning with the coniin phenomena analogous to those of curare.

Coniin, in a condition of absolute purity, acts on the cerebro-spinal centres; it is neither a muscular nor a cardiac poison. The first effect is a general enfeeblement, then convulsive shuddering; these phenomena are followed by a period of augmentation of reflex excitability, while at the same time voluntary movements are abolished, and the respiration is accelerated; visual troubles are also noted. In a more advanced stage of the intoxication, the reflex excitability gradually disappears, the respiration and pulse are simultaneously weakened; then follows a profound collapse, which may not be succeeded by death.

The chloro-hydrate and bromo-hydrate of conicine (Mourrut) give rise to the same phenomena of poisoning as conia; they seem to be even more energetic. Conicine has a very irritant local action; when injected under the skin it produces sphacelus, and is not entirely absorbed.

M. Tiryakian finishes his study by seeking to discover the physiological and medicinal effects of bromo-hydrate of coniin on man. To obtain a notable effect, it should be administered in a dose of 0.1 gramme at least, in a single dose for an adult; this may be increased to the amount of one gramme *per diem*, either internally or injected hypodermically (the bromo-hydrate not being irritant); thus showing that the toxic action of coniin has been much exaggerated.

The author has experimented with the bromo-hydrate of coniin in affections accompanied with cough, in epilepsy, in neuralgias, but is not yet able to say precisely what is the therapeutic action of the drug.

GEISSINE.—This substance, the alkaloid of pao-pereira has been investigated by MM. Bochefontaine and Cypriano de Freitas, *Gaz. Méd. de Paris*, 1878, Nos. 16 and 17. The following are their conclusions as reported in *Revue des Sci. Médicales:*

1. Geissine is a paralysant poison. It causes a progressive enfeeblement of all the movements, whether voluntary or reflexes of animal life, and next a loss of sensibility.

2. The paralysis is not due to an action of the poison on the muscles nor on the peripheral nerves.

3. The toxic action affects the nerve centres, especially the spinal cord and medulla, the functions of which are progressively abolished.

4. In some of the author's experiments there was noticed a diminution in the number of cardiac pulsations, which was also remarked by José Silva and Gonsalvez Ramos.

THE TREATMENT OF INSANITY BY DRUGS.—Dr. Geo. H. Savage, in the last volume of Guy's Hospital Reports, has contributed an article on this subject, the main points of which are given in the *Dublin Medical Press* and *Canada Medical Record* for December. We take them from the latter journal:

Until quite recently, observes Dr. Savage, *opiates* were looked upon as one of the sheet-anchors in the arrest of mental disease. Now we are more discriminating, and have to own that, whereas some cases are relieved by opium, some are not affected at all, or are really injured by its use. In the first place, the effect of this drug will vary with its mode of administration. Some cases are not improved by morphia administered by the mouth, but will recover, or be greatly benefited, by the subcutaneous injection of that alkaloid. Two or three cases are reported where no improvement took place until the patient was put on a solution of morphia, in half-grain doses, two or three times a day, when a decided change for the better took place, and even ultimate recovery. Another case showed how morphia will control symptoms, though it may be long before it perfects a cure: and in the author's experience, "when symptoms are so controlled it is only a question of time to cure." Another patient with active melancholia, was quiet and happy so long as she took morphia, but if this was discontinued she became very irritable. In her case no medical treatment had been tried for two months previously to the administration of the morphia, and within twenty-four hours from the commencement of this drug she became quiet and reasonable. She is still under treatment but will recover. In short, Dr. Savage would say that morphia has served him well in active melancholia both in old and young, but especially in old cases, such as climacteric and senile patients: also where sleeplessness alone seems the cause of the mental breakdown, and in some cases of excitement in which chloral-taking or over stimulation has caused insanity; but it is of no avail in ordinary acute mania, general paralysis, profound melancholia, or complete dementia.

With regard to chloral hydrate the writer would restrict its use to only a few forms of insanity. He justly remarks that "of all medicines recently introduced this has been the most largely used, and I fear if the good results were compared with the evil done the latter would preponderate." The mere producing of sleep does little, if any, good in the majority of cases of insanity. It is, however, useful in the epileptic states, in the furore of epilepsy, and in some cases of insanity from excess of stimulants. In one case, where there was furious mania following epileptic fits, the chloral was sometimes given, and the results were always quietness with chloral and mania without.

Dr. Savage also speaks in favor of a combination of chloral and camphor (10 grains of each, rubbed up with simple syrup), which was especially tried in two classes of cases—the wildly and distinctly maniacal—who were filthy in their habits, and in those who were erotic or lascivious in their behavior. The mixture produced a good effect; and out of twenty cases in which it was given, fourteen were made more quiet. The use of camphor, moreover, obviated the loss of appetite and of flesh, which was produced by prolonged use of chloral alone, and all the patients gained in weight and improved in appetite. In more than one case the patient was quiet and decent while taking the medicine, and one case had every appearance of becoming a chronic lunatic, until the chloral and camphor were given. The writer would recommend this combination in cases of puerperal insanity, especially in the sleepless chattering form, where friends are mistaken, and erotic feelings are present.

Of the value of conium the report is not very encouraging: In a case of violent mania it was of some benefit after injection of morphia, camphor and chloral, and other remedies had failed; and it is recommended in cases where patients are noisy and destructive, but, at the same time, require stimulants.

Of still less value is hyoscyamine, the effects of which are so powerful and dangerous that sickness and collapse have been known to follow one dose of it. In one case a thirteenth of a grain produced in an hour and a half complete inability to stand, sickness, cold, clammy skin, and absence of radial pulsation, without any good results following.

Of bromide of potassium the author has not a good opinion, but he confesses that his experience of that drug has not been very great.

Of all medicines *purgatives* have been most favorable with the older physicians and the majority of the best writers on insanity. But Dr. Savage says, "we rarely give them at Bethlem with the idea that we shall cure by these means, and still more rarely to quiet the patient and keep him employed." *Stimulants*, on the other hand, are more favorably spoken of. We are told that stimulants are a large item in the expenditure of asylums, and, when judiciously ordered and watched, they are of the utmost importance. Emmenagogues were also found of great service in the treatment of insanity, complicated with amenorrhœa. Of this class of drugs the tincture of *black hellebore*, in doses of half a drachm to a drachm, was remarkably beneficial, and several cases are cited in which both the amenorrhœa and insanity yielded to this remedy. The re-establishment of menstruation is important, and the return of menstruation unaccompanied by a mental change, adds to the gravity of the prognosis.

Independently of ordering medicinal remedies, there are certain physical conditions which often contribute to the cure of insanity, and Dr. Savage draws particular attention to cases of this disease, in which physical illness produced marked improvement in the mind of the patient. Thus several forms of insanity respectively, got well spontaneously after the formation of a retro-uterine hæmatocele, after a toothache and gum-boil, after inflammation of lower jaw, after an attack of erysipelas of head, after obstruction of the bowels, and after an attack of gout. Dr. Savage does not draw any inferences from these circumstances, but we should think that the good result often following distant irritation in the form of a natural disease might suggest the propriety of resorting to counter-irritation in the treatment of insanity more frequently than we do now. "In former times the head-shaving and blistering treatment must certainly have improved some cases, just as we have found that in some, purgatives are beneficial." (*Dublin Medical Press*, Oct. 2, 1878.) (*Canada Medical Record*, November, 1878.)

The following are the titles of some of the recently published papers on the Therapeutics of the Nervous System and Mind:

Mills, Massage and Swedish Movements in the Treatment of Diseases of the Nervous System, *Med. and Surg. Rep.*, Oct. 5.—Mackay, On the Value of Spiritus Nucis Juglandis in the Treatment of Vomiting, *Practitioner*, Dec.—Pallen, Hypodermics of Coffee for Morphia Vomitings and Opium Poisoning, *N. Y. Med. Record*, Dec. 21.—Turnbull, Ether *vs.* Chloroform, Bromide of Ethyl, *Va. Med. Monthly*, Dec.—Renton, On Bromide of Potassium in Epilepsy, *Glasgow Med. Journal*, Dec.—Voisin, Study of the Treatment of General Paralysis of the Insane, *Bull. Gen. de Thérap.*, Dec. 15.

BOOKS, ETC., RECEIVED.

Structure des Circonvolutions Cérèbrales (Anatomie et Physiologie). Thèse présenteé et Soutenue a la Faculté de Médecine de Paris, pour le Concours de l'Agregation. Par le Dr. Charles Richet. Paris, 1878. 172 pages.

Les Terminaisons des Nerfs dans la Peau. Thèse présenteé et Soutenue a la Faculté de Médecine de Paris. Par le Dr. Louis Couty. Paris, 1878. 172 pages.

Handbuch der Speciellen Pathologie und Therapie. Herausgegeben von Dr. H. v. Ziemssen. Achter Band. Krankheiten des Chylopoëtischen Apparates. II.—Erste Hælfte, Erste Abtheilung, Leberkrankheiten. Von Prof. E. Ponfick, Prof. Dr. Thierfelder, Prof. O. Schueppel, Prof. O. Leichtenstern und Prof. A. Heller. 440 pages.

Elfter Band. Zweite Haelfte. Krankheiten des Rueckenmarks. Dritte (Schluss.) Abtheilung. Von. Prof. Wilhelm Erb in Heidelberg. Mit 12 Holzschnitten. 1878. 141 pages. Supplement Band. 1878. 380 pages.

On Rest and Pain: A Course of Lectures on the Influence of Mechanical and Physiological Rest in the Treatment of Accidents and Surgical Diseases, and the Diagnostic Value of Pain. Delivered at the Royal College of Surgeons of England, in the years 1860, 1861 and 1862. By John Hilton, F. R.S., F.R.C.S. Edited by W. H. A. Jacobson, F.R.C.S. Second Edition. New York: Wm. Wood & Co. 1879. 299 pages. Chicago: Jansen, McClurg & Co.

Die Geisteskræfte der Menschen verglichen mit denen der Thiere Ein Bedenken gegen Darwin's Ansicht ueber denselber Gegenstand. Von Ludwig Struempell. Leipzig, 1878. 64 pages.

Essentials of Chemistry, Inorganic and Organic. For the Use of Students in Medicine. By R. A. Witthaus, A. M., M. D. New York: Wm. Wood & Co. 1879. 257 pages. Chicago: Jansen, McClurg & Co.

Notes on the Treatment of Skin Diseases. By Robert Liveing, A. M., M. D., Cantab, Fourth Edition, revised and enlarged. New York: Wm. Wood & Co., 1878. 123 pages. Chicago: Jansen, McClurg & Co.

Studien ueber den Nervus Vagus. Ein Beitrag zur Lehre von der Automatischen Nervencentren und den Hemmungsnerven. Von Dr. Ottomar Rosenbach. Berlin, 1877. 140 pages.

Diseases of the Bladder and Urethra in Women. By Alexander J. C. Skene, M. D. New York: William Wood & Co., 1878. 374 pages. Chicago: Jansen, McClurg & Co.

A Manual of Physical Diagnosis. By Francis Delafield, M. D., and Chas. F. Stillman, M. D. New York: William Wood & Co., 1878. Chicago: Jansen, McClurg & Co.

A Manual of Prescription Writing. With a full Explanation of the Methods of Correctly Writing Prescriptions; A Table of Doses, expressed in both the Apothecaries' and Metric Systems; Rules for Avoiding Incompatibilities and for Combining Medicines. By Matthew D. Mann. New York: G. P. Putnam's Sons. 155 pages. Chicago: Jansen, McClurg & Co.

Transactions of the Twenty-eighth Anniversary Meeting of the Illinois State Medical Society. Held in the City of Springfield, May 21, 22, 1878. Chicago, 1878. 275 pages.

Transactions of the Pathological Society of Philadelphia. Volume Seventh. Containing the Report of the Proceedings for the Sessions from September, 1876, to July, 1877. Philadelphia, 1878. Printed for the Society by J. B. Lippincott & Co. Chicago: Jansen, McClurg & Co.

The Annual Report of the Board of Regents of the Smithsonian Institution. Showing the Operations, Expenditures, and Condition of the Institution for the year 1877. Washington, 1878.

Lectures on Bright's Diseases of the Kidneys. Delivered at the School of Medicine, of Paris, by J. M. Charcot. Collected and Published by Drs. Bourneville and Sevestre, Editors of the *Progrès Médical*, and Translated, with the permission of the Author, by Henry B. Millard, M. D., A. M. New York: William Wood & Co., 1878. 100 pages. Chicago: Jansen, McClurg & Co.

Studies in Pathological Anatomy. By Francis Delafield, M. D. Nos. 3 and 4, April and May, 1878. New York: William Wood & Co., 1878.

Harvey and his Discovery. By J. M. DaCosta, M. D., Professor of the Practice of Medicine, at the Jefferson Medical College, Philadelphia. Philadelphia, 1878: J. B. Lippincott & Co. Chicago [illegible] a [illegible] urg & Co.

On the Use of the Solid Rubber Bandage in the Treatment of Eczema and Ulcers of the Leg. By L. Duncan Bulkley, A. M., M. D. (Reprinted from the *Archives of Dermatology*, July, 1878.)

Pocket Therapeutics and Dose Book, with Classification and Explanation of the Action of Medicines, &c., &c. By Morse Stewart, Jr., B. A., M. D. Detroit, Mich., 1878. 101 pages.

Treatment of Fracture of the Shaft of the Femur. The "American Method." A Paper read before the Minnesota State Medical Society, at St. Peter, June 18th, 1878. By Franklin Staples, M. D., of Winona. (Reprinted from the Transactions of the Society.)

Report on the Care of the Insane. By John Curwen, M. D., of Harrisburg. (Extracted from the Transactions of the Medical Society of the State of Pennsylvania.)

Ueber Einen Fall von Reflexschwindel aus bisher nicht beschriebener Ursache. Von Dr. Med. A. Erlenmeyer. (Separatabdruck aus der *Deutschen Medicinischen Wochenschrift*, Nos. 44 and 45. 1878. (See Periscope of this issue.)

Morbid Histology of the Spinal Cord in Five Cases of Insanity. By Rengrose Atkins, M. A., M. D. (Reprinted from the *British Medical Journal*, 1878.)

Pathological Illustrations of the Localization of the Motor Functions of the Brain. By Rengrose Atkins, M. A., M. D. (Reprinted from the *Brit. Med. Journ.*, May 4 and 11, 1878. (These two papers are useful contributions to neurological literature by a British alienist, who is a neurologist as well.)

Address of Joseph K. Edgerton, President of the Board of Trustees of the Fort Wayne Medical College, to the Faculty and Students, at Fort Wayne, Ind., Oct. 1st, 1878.

The Constituents of Climate, with Special Reference to the Climate of Florida. By Frederick D. Lente, A. M., M. D. (From August No. *Richmond and Louisville Medical Journal.*) Louisville, Ky., 1878.

Handbuch der Geistes-Krankheiten. Von Dr. H. Schuele, in Illenau. Leipzig, 1878.

Carbolic Acid Injections in the Treatment of Piles. Radical Cures. By A. B. Cook, A. M., M. D. (From the *Am. Med. Bi-Weekly*, Feb. 16, 1878.)

The Duties of the Medical Profession Concerning Prostitution and its Allied Vices. Being the Oration before the Maine Medical Association at its annual meeting, 12th of June, 1878. By Frederick Henry Gerrish, M. D. (More remarkable for its extreme views than for its value).

A Case of Acute Puerperal Inversion of the Uterus. A Clinical Description of a new Instrument Successfully Employed. With Remarks on the Mechanism of Restoration. By John Byrne, M. D., M.R.C.S.E. (Reprinted from the *New York Med. Jour.*, Oct. and Dec., 1878.)

Annual Address Delivered before the American Academy of Medicine, at Easton, Pa., Sept. 17, 1878. By Frank H. Hamilton, A. M., M. D., LL. D. of New York. Published by order of the Academy.

Laceration of the Cervix Uteri.. The Address in Obstetrics Delivered before the Medical Society of the State of Pennsylvania, by William Goodell, A. M., M. D., at its Annual Meeting held at Pittsburg, May, 1878. (Reprinted from its Transactions.)

The Present State of Otology. By S. J. Jones, A. M., M. D. (Extracted from the Transactions of the Illinois State Medical Society, Springfield, May, 1878.)

Affections of the Lachrymal Apparatus. By S. J. Jones, A. M., M.D. (Extracted from the Transactions of the Illinois State Medical Society, Springfield, May, 1878.)

Inversion of the Bladder. By Reuben A. Vance, M. D., of Gallipolis, Ohio. (Extr. from the *Am. Journal of Med. Sciences*, for October, 1878.)

Report of the Special Commissioners Appointed under the Provisions of Joint Resolution No. 137, entitled, a "Joint Resolution in Relation to the Insane of the State and the Statutes in Relation to their Confinement and Treatment," approved November 28th, 1876. Presented by M. Goldsmith of Rutland, O. F. Fassett of St. Albans, W. H. Walker of Ludlow, Commissioners, October, 1878. Rutland, Vt., 1878. (With Plans of State Hospital for Insane, Brattleboro, Vt.)

The Local Treatment of Eczema. By Henry G. Piffard, M. D. (Rep. from *The Medical Record*, Oct. 26, 1878.)

Restoration of the Membrana Tympani. By S. O. Richey, M. D. (Extr. from the Transactions of the Illinois State Medical Society, Springfield, May, 1878.)

On Diet and Hygiene in Diseases of the Skin. By L. Duncan Bulkley, A. M., M. D. (Reprinted from the *Virginia Med. Monthly*, Oct., 1878.)

Fourth Annual Report of the Officers and Superintendent of the Asylum at Walnut Hill, Hartford, Conn., at their Annual Meeting, October, 9, 1878. Also Petition to the Legislature. Hartford, 1878.

Twenty-third Annual Report of the Trustees of the State Lunatic Hospital at Northampton, for the Year Ending September 30, 1878.

Annual Report of the Trustees and Superintendent of the State Lunatic Hospital of Pennsylvania. 1878.

Ninety-sixth Annual Catalogue of the Medical School (Boston) of Harvard University, 1878-9. (Reprinted from the Catalogue of the University.)

THE FOLLOWING FOREIGN PERIODICALS HAVE BEEN RECEIVED SINCE OUR LAST ISSUE.

Allgemeine Zeitschrift fuer Psychiatrie und Psychisch. Gerichtl. Medicin.
Annales Médico-Psychologiques.
Archiv fuer Anatomie, Physiologie, und Wissenschaftl. Medicin.
Archiv fuer Path. Anatomie, Physiologie, und fuer Klin. Medicin.
Archiv fuer die Gesammte Physiologie der Menschen und Thiere.
Australian Practitioner.
Brain.
British Medical Journal.
Bulletin Générale de Thérapeutique.
Centralblatt f. d. Med. Wissenschaften.
Centralblatt f. d. Nervenheilk., Psychiatrie, etc.
Cronica Med. Quirurg. de la Habana.
Dublin Journal of Medicine and Surgery.
Deutsche Medicinische Wochenschrift.
Edinburgh Medical Journal.
Gazetta Frenocomia di Reggio.
Gazetta Medica de Roma.
Gazette des Hopitaux.
Gazette Médicale de Bordeaux.
Giornale Internazionale della Scienze Medicale.
Glasgow Medical Journal.
Hygeia.
Hospitals Tidende.
Journal de Médicine et de Chirurgie Pratiques.
Journal of Mental Science.
Journal of Psychological Medicine.
Journal de Medecine de Bordeaux.
La France Médicale.
Lancet.
Le Progrès Medical.
Lo Sperimentale.
L'Union Medicale.
Mind.
Nordiskt Medicinskt Arkiv.
Norsk Magazin for Lagensvidenskabens.
Psychiatrisches Centralblatt.
Rivista Clinica di Bologna.
Rivista Sperimentale di Freniatria e de Medicina Legale.
Revue Medicale du Nord-Est
Revue Mensuelle de Medicine et de Chirurgie.

Schmidt's Jahrbuecher der In- und Auslandischen Gesammten Medicin.
St. Petersburger Med. Wochenschrift.
The Practitioner.
Upsala Lakareforenings Forehandlinger.

The following domestic exchanges have been received:

American Journal of Insanity.
American Journal of Medical Sciences.
American Journal of Obstetrics.
American Journal of Pharmacy.
American Medical Review and Index.
American Medical Weekly.
American Practitioner.
Archives of Dermatology.
Atlanta Medical and Surgical Journal.
Boston Medical and Surgical Journal.
Buffalo Medical Journal.
Canada Medical Record.
Canadian Journal of Medical Sciences.
Chicago Medical Journal and Examiner.
Clinic.
Cincinnati Lancet and Observer.
Detroit Lancet.
Maryland Medical Journal.
Medical Brief.
Medical News and Library.
Medical Record.
Medical and Surgical Reporter.
Michigan Medical News.
Nashville Journal of Medicine.
New Remedies.
New York Medical Journal.
New York Med. and Surg. Brief.
Ohio Medical and Surgical Journal.
Pacific Medical and Surgical Journal.
Proceedings of Med. Society of the County of Kings, N. Y.
Pharmacist.
Philadelphia Medical Times.
Quarterly Journal of Inebriety.
Richmond and Louisville Medical Journal.
Southern Practitioner.
St. Louis Medical and Surgical Journal.
St. Louis Clinical Record.
Toledo Medical Journal.
Virginia Medical Monthly.

THE
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Original Articles, Selections and Translations.

Art. I.—THE ANATOMICAL AND PHYSIOLOGICAL EFFECTS OF STRYCHNIA ON THE BRAIN, SPINAL CORD AND NERVES.

By Edward C. Spitzka, M. D.,

Professor of Comparative Anatomy, and Lecturer on Nervous Diseases, in the Columbia Veterinary College, Vice-President of the New York Neurological Society, Curator and Pathologist to the New York Medico-Legal Society, Member of the American Neurological Association, W. and S. Tuke Prize Essayist, Etc., Etc.

[Being the essay to which the Wm. A. Hammond prize of $250 was awarded by the Committee of the American Neurological Association, at their session of 1878.]

IN an essay of the character of the present one, it is neither desirable nor necessary that equal prominence should be given to each individual question involved in the theme. Many points relating to the subject of Strychnia poisoning may be regarded as satisfactorily settled, and further than that the writer has recorded the fact that his experiments have verified the well established *dicta* of previous writers, he has devoted but little space to them.

Except in so far as it has been deemed essential to a fair contrasting of the effects of Strychnia on different classes of animals, or of different doses on species of the same class, I have not specially detailed those phenomena observed on the administration of this alkaloid, which are familiar to all. The chief interest in these phenomena, certainly does not centre in the fact that there are convulsions, *et cetera*, but in the problems which the latter suggest to the physiological logician.

Among such problems, the following are the most prominent:

I. Is there any one portion of the nervous system on which strychnia localizes, or concentrates its action?

II. If such a predilection for a special division of the nervous system exists, is the *locus predilictionis* the same in all classes of vertebrates?

III. Does strychnia produce its peculiar motor influence in any way other than the one usually accorded it, of increasing the reflex excitability?

IV. Does strychnia induce any essential pathological changes?

V. Does the action of the agents employed experimentally or therapeutically as antidotes to strychnia, depend upon a true antidotal influence (chemical antithesis), or merely on their virtue in annulling certain *results* of the poisonous action?

VI. Having determined the anatomical and physiological effects of acute strychnia poisoning, it remains for us to study what practically, at least, is of equal importance: the results of chronic strychnia poisoning.

Each of the foregoing questions involves other subsidiary ones, which will be considered in their proper connection. I have ranged the various experiments and their discussion under the head of propositions embodying the results obtained.

As the effects of strychnia are not uniform at different temperatures, it may be well to state that the experiments demonstrating the different actions of varying doses, were performed in winter, in an unheated room, whose temperature ranged from 2° to 15° C.

Unless otherwise specified, the preparation of strychnia employed has been the acetate.

PROPOSITION I.

The more the central nervous system preponderates over the scattered ganglionic centres, the more pronounced are the exalted actions of the voluntary muscles. Among vertebrates the higher we rise in the scale, the more pronounced do clonic convulsions become, as contrasted with tonic convulsions, and the more apt are we to find intervals of apparent recovery from the strychnia effects occurring. The rate of the clonic spasms differs according to the rate of rapidity of the motions of a given species under natural circumstances.

In order to compare the effects of the alkaloid on different species, I have performed a series of preliminary experiments on such animals as were accessible to me, grading the dose proportionately to the weight of the animal. Such dose gradation can be carried out with those animals only which are air-breathers, or with such water-breathers as permit of the application of the hypodermic method. With other aquatic species, the poison has been added to their respiratory medium.

I append a list of the animals experimented on, as well as of their number:

1.	Equus caballus (horse)	1
2.	Canis familiaris (dog) adult	6
2.	Canis familiaris (dog) puppies	11
3.	Felis catus (cat) adult	4
3.	Felis catus (cat) kittens	10
4.	Capra hircus (goat)	1
4.	Lepus cuniculus (rabbit)	9
6.	Cavia cobaya (guinea pig)	2
7.	Vespertilio Noveboracensis (bat)	3
8.	Mus musculus	1
9.	Didelphys virginiana (opossum)	1
10.	Corvus Americanus (crow)	1
11.	Columba (pigeon)	2
12.	Alligator Mississipiensis	2
13.	Chelydra serpentina (snapping turtle)	1
14.	Chrysemys picta (painted turtle)	2
15.	Malacoclemmys palustris (terrapin)	1

16.	Nanemys guttata (sp. turtle)	3
17.	Cistudo Carolina (box-turtle)	2
18.	Rana pipiens (bull-frog)	1
19.	Rana leopardina (banded frog)	255
20.	Rana fontinalis (green frog)	10
21.	Rana halecina (shad frog)	62
22.	Bufo Americanus (common toad)	420
23.	Salamander erythronotus	6
24.	Triton	2
26.	Cyprinus aureus (gold fish)	1
27.	Salmo salar (salmon fry)	17
28.	Gasterosteus Noveboracencis (stickleback)	6
29.	Embryo of common barn fowl	4
30.	Embryo of California salmon	32
31.	Embryo of skates (Raia lævis, aculeatus)	3
32.	A large number of mollusks and microscopic organisms.	

Experiment 29.—An animalcula stage was filled with water containing numerous rotifers, keronias, and paramecium, with some vampyrellas and monads. After noting that all the animals were either actively swimming about, or that at least their cilia were acting vigorously, three-tenths of a milligramme of strychnia acetas were added. Almost simultaneously the vampyrellas in the field ceased moving, the rhythmical contractions of the vesicles in paramecium became arrested, and other species floated dead, and shrivelled or collapsed across the field. The rotifera held out longer, some of them manifesting an increased rapidity of pedicular contractions at the outset, but this may have been due to the agitation produced by the perturbation of the fluid; within thirty seconds they were quiescent and *relaxed*. Several protomonas were still swimming about at this time, but much more slowly than before, and at the end of four minutes not a single living specimen could be found. What is noteworthy in this experiment is that none of the species showed the slightest tetanic symptom. That the acid condition of the added solution might not be accused of having caused their death, I have added to a similar specimen of water an equally acid solution of glacial acetic acid; the vampyrellas especially survived a long time

in this solution, and not even the delicate keronias died before an hour or more had elapsed.

Experiment 281.—A specimen of the microscopic water crustacean, Cyclops quadricornis, was placed in the animalcula stage. As usual after transferral, the animal darted around with its characteristic abrupt dashes, then when it became quiescent, .0001 of S. was added to the water; the commotion caused by the addition of the new fluid caused it to start and dart about as normally, but at the end of twenty seconds, its dashes became shorter, as if checked half way; this gradually increased, the movements remaining as frequent, but the distance made by each movement becoming shorter and shorter, until the animal became perfectly motionless. Under the microscope, its antennæ were found stiffly extended, its caudal spines and natatory members erect, the heart was quiescent, except for a faint and almost imperceptible contraction at the end of two minutes. At the end of 2′ 40″ from the time the poisonous solution was added, the animal was relaxed, becoming opaque, a sign that it was dead.

Experiments 224, 225, 226, 227.—Four sticklebacks were placed in a large glass globe containing about one-half a gallon of sea-water, and in a well-lighted room; it was remarked that three were females, and one a male, arrayed in the crimson tints peculiar to the male stickleback at breeding time. Twelve grains (0.75) of S. were added to the water. Twenty minutes elapsed before any effect was noticed; this manifested itself by the fact that the slightest jar to the table on which the globe was standing would cause the animals to start and dart about much more rapidly, than other sticklebacks in non-strychninized water placed on the same table. Twenty-five minutes after administration, their movements became more jerking in character, and five minutes later, the females began to attack each other fiercely, and continued doing so repeatedly for a quarter of an hour. Previous to and during this stage the pectoral fins of all four individuals were moving with nearly twice the rapidity of the normal rate. This movement was clearly a respiratory one, and had for its purpose the increase in rapidity of the water current flowing past the gills. It rose and sank parallel with the functionally associated oral movements, and the animals were not propelled through the water by these fin movements; the caudal fins were motionless, or nearly so.

Seventy minutes from the time of administration, two of the females were seen lying at the bottom of the globe, their ventral and dorsal spines *permanently* erect, and so continuing until death. At intervals of five minutes the other two fish passed into the same state, the male last, and without losing his color. The rigidity of this erection, and of the entire muscular system, was so great that the previously agile and excitable fish could be seized by one of the dorsal spines, without making any attempt at escaping, and lifted out; they could be held out by the tail, as stiff as rods, and could be laid on the table without making any, even the slightest, struggle. While in the water, the only vital manifestations retained, were the color and the movements of the mouth, which seemed to be drawn into extreme extension at times. The previously increased pectoral fin movements were now abolished, the fins being in extreme erection.

At this stage, one of the females, whose oral movements had ceased, was opened, the viscera were found pale, and the heart *empty*, *contracted*, and motionless. On returning the animal to the water, its oral motions *returned*, and continued faintly and at long intervals for four minutes after, the heart remaining as before. The moment of dying was marked by a gradual and general paling of the natural tints of the body, especially of the sclerotic zone. Death took place in the four animals as follows, from the time of administration:

1. Female, opened as stated, 1 hour, 42 minutes.
2. Female, frequently disturbed, 1 hour, 47 minutes.
3. Female, undisturbed, 1 hour, 55 minutes.
4. Male, 2 hours, 6 minutes.

The significance of this experiment lies in the fact, that one complicating factor, present in higher animals, could be here, at least partially excluded. In air-breathers the initial spasms affecting the respiratory muscles, in conjunction with the entire motor apparatus, cause a partial asphyxia, which as I shall show, involves many consequences not primarily attributable to the strychnia.

Here, however, we had no asphyxia. All cavities of the heart were found empty, and the injection of the gills was normal in quantity and color. This immunity is referable to the fact that on account of the incompressible walls of the gill cavity, muscular spasm cannot affect the latter by pressure. The fish passed into their final tonic spasm before even a suspicion of asphyxia could be justified.* It might appear perhaps that the increased (respiratory) movements of the mouth and pectoral fins signified a respiratory need, but as the same movements are found equally increased in non-strychninized, *excited* sticklebacks, and as further in the strychninized specimens, increased pugnacity and general excitability co-existed at that stage, I look upon it as a psychical symptom. The salient feature in the tetanus of these animals is, that it was *tonic* throughout, not being interrupted by clonic spasms at any time.

Experiments 48 and 122.—A young alligator thirteen inches in length had 0.0075 S. injected into his peritoneal cavity. In four minutes he became more restless, and struck around with his tail; his respiratory movements became more excursive, and at 4′ 30″, his head was violently drawn back, while a slight extensor wave ran down the vertebral column. From this the alligator recovered by a visible effort, and in subsequent spasms, this struggle between the effects of the poison and the efforts of the will, could be observed—in this respect presenting a considerable contrast to the frog, who submits readily to the tetanizing influences. At intervals of from two to four minutes, the alligator had distinct spasms like the first, and in the intervals would open his jaws widely, as if to rid himself of an incipient trismus. Trismus of an intense character was a marked feature throughout, and the pupils were *contracted.* At 16′ the animal passed into a continuous opisthotonos, to which frequent successive clonic shocks were superadded.

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*That the cessation of the fin movements might not be accused of having produced asphyxia, and thus the death of the animal, during the terminal tetanus (since upon these movements depends the continuance of the branchial current) I may state that the stickleback can be kept alive for days when the pectoral fins are amputated. This fish is perhaps the hardiest denizen of our waters, and is a fitter subject for experimentation than the delicate gold-fish, usually selected.

After the spasm had lasted eight minutes, the animal being rigid and motionless (and the pupil still moderately contracted), it was opened, while the viscera were successively exposed and manipulated, the violent tetanus continued, the jaws could be heard to grate against each other, and the limbs were extended along the sides. The viscera were found pale, the lungs moderately hyperæmic, heart beating infrequently, irregularly and checked in systole, hemispheres and oblongata injected, cord normal.

Another alligator, length 18″, was poisoned *per orem*, by 0.015 S. The first spasm took place 12′ after administration, death in 1 hr. 10′. The pupils became dilated moderately before death, the lungs were found congested, the medulla and cord hyperæmic, hemispheres moderately injected.

Experiment —*An opossum received 0.25 S. *per orem*; for eighteen minutes nothing unusual was noticed, except that an attempt to "hypnotize" the animal, (which under normal circumstances is easy,) failed. Then an occasional stiffness in gait, varied by clonic spasms of the fleshy panniculus, alternating with free intervals, occurred, until 32′ from the time of administration, when a marked opisthotonos took place, lasting, with variations in degree, but interrupted by complete relaxation, until death, which occurred twenty-two minutes later. The animal never regained its feet, for the hind legs, besides being extended, were adducted, so that the claws touched each other. At intervals of a minute violent clonic spasms, lasting from two to five minutes at a time, occurred; the end of the last clonic spasm left the animal supple and relaxed, marking its death. Autopsy: all the thoracic and abdominal viscera congested, the right side of the heart moderately filled with dark blood; on pinching the heart a few feeble contractions were provoked; brain and cord hyperæmic, a large clot in the *tela choroidea*, and over the optic lobes and trapezium, color of the effusions purplish.

Experiment 1.—A large starved cat received eight grains of acetate of strychnia (0.48) wrapped up in meat, which was sewed together. A subsequent examination showed that none of the strychnia could have remained in the mouth. The symptoms are recorded in their order, counting from the time of administration. It may be stated that the dose was given in three instalments, each wrapped up as stated, was swallowed ravenously; the administration extended over two minutes. 3′, the ears twitch at the slightest sound, or even without any appreciable cause, the pupils contract and dilate under stimulus, naturally, but there is a tendency to dilatation; 3′ 20″, horripilations; 3′ 45″, it starts, as a cat would do, in pursuit, but suddenly, as if frightened, it jumps on a chair; horripilations continue; 5′, the right paw starts from its position, involuntarily without doubt, for the cat recovers its position by a manifest will-effort; 5′ 5″, the same paw starts out again horizontally, and is agitated by a genuine *epileptiform* spasm, the rest of the body, except for the movements of the hairs, being entirely quiet. This spasm was unmistakably like those produced by localized cortical electrization, and lasted 6′, the animal recovering itself as before; 7′ 8″, respirations twenty-six per minute, and not labored; previous to administration they were eighteen; 8′, it licks its mouth and acts as if it had an unpleasant taste therein; 9′, starts as if to jump through the crack of the half-open door through which I am watching it, but a stiffening of the flexors of the hind legs prevents it from carrying this out; 11′, with a violent effort, it jumps for the desired egress, but stiffens out, and turns over completely in mid-air, falling on the ground in extreme opisthotonos. For twenty seconds, general tonic and clonic spasms of the most frightfully intense character (during which the animal split one of its molars) agitated the animal: at the end of that time its pupils were found to be extremely dilated, the animal supple, death. Autopsy: brain and cord moderately injected (venous).

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*Not in essay as originally presented.

Experiment 2.—Full grown cat, given fifteen grains in same manner as in preceding case. The notes are fuller; I select from them the observation that the attention of the animal for the surroundings was heightened, that the diameter of the pupils reached its maximum when the spasms were at their height, and that an *epileptiform* spasm of the right ear was noticed similar to the spasm of the fore-leg noticed in experiment 1. Death occurred in nine seconds from the commencement of the only and terminal tetanic convulsion. The autopsy made immediately after death,* revealed complete anæmia of the brain and cord, heart contracted in systole; right side moderately filled with dark blood, great veins moderately filled with lake-colored blood, lungs pale.

In these experiments very large doses were given, in order that the initial symptoms should be sufficiently well marked to become objectively noticeable, it being well known that these initial symptoms (horripilations, psychical inquietude, *et cetera*), are but imperfectly developed, or even absent when the poison is given in small doses, or in minimal doses often repeated.

In the lowest animal forms, we perceive that strychnia causes death by checking the protoplasmic movements (cilia) without causing either clonic or tonic spasms. The movements of ciliated epithelium, of white blood corpuscles, and of spermatozoa, are checked in like manner. The higher rotifera and crustacea already exhibit the tonic spasm coming on gradually and terminating life without any preliminary excitement. The fish dies in the same manner, but although he does not exhibit other than tonic spasms, he shows an acceleration of the respiratory movements and increased psychical irritability as manifested in the attacks on other fish. The caudate batrachia present the same phenomena, but an occasional tremor foreshadows the well-developed clonic spasm characteristic of the caudate amphibians. The alligator, representing the highest group of existing reptiles, already manifests the combination of well-developed clonic and tonic spasms; found with mammals, but there are no intermissions. In the opossum there are intermissions of the clonic but not of the tonic spasms, in the dog and human being complete intermissions are found. These remarks I have stated apply to cases where larger doses are given, and exclude those instances in which the first spasm was at the same time terminal and fatal as in experiments 1 and 2. But in order to exclude any source of error arising from the largeness of the dose employed, I have experimented on a similar series with minimal doses, and found that if strychnia has any influence on the protozoa, at all, it kills them without producing any previous spasms; that it kills most *crustaceans* and some mollusks in tonic spasms, that fish die in the same condition, or if they survive have no recurrence of the same, that frogs, alligators and turtles either die in a mixed tonic and clonic convulsion, or if they recover from the first attack, may have a recurrence, which is always milder than the original attack, and that the mammals with minimal lethal doses, have frequent intervals, and that the succeeding spasms are not different in intensity from the first one.

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*Since writing the foregoing I have found that in order to determine the degree of injection of the cerebral vessels, it is unwise to perform the autopsy immediately after death, as then the blood, still fluid, runs from the large veins of the membrana obturatoria and the emissaries, rendering the record valueless.

In the course of these same experiments, I have found that where clonic convulsions occur, their special character depends upon the special development of the muscular periphery in a given species. In the frog and toad the clonic spasms resemble exaggerated natatorial movements, or jumps, in the turtle the powerful retractors and protractors of the head are in continual action, the head shooting out and in of the shell, and to this are added in the box-tortoise corresponding movements of the plastron. It often happens in the latter species that the limbs being protruded during a clonic spasm, are caught and pinched by the lid closing in a tonic one.* This latter instance shows that at one and the same time, one group of muscles may be in tonic and others in clonic spasms, under the influence of this alkaloid.

There is no essential difference between the herbivorous and carnivorous species, either in regard to their vulnerability to the alkaloid, or the intensity of their motor reaction.

It is a current statement in works on experimental physiology that the frog is peculiarly susceptible to the influence of strychnia. I believe this to be a fallacy, and adduce the following experiments to exhibit it:

Experiment 6—Large *Rana fontinalis*, weight 130 grammes, 0.0075 strychnia injected hypodermically. In 18″ the movements of the nasal orifice became accelerated, and the flank movements gradually increased until the animal became tetanic, agitated by frequent clonic spasms at the end of 6′. These spasms reached their maximum in one hour, and becoming weaker and less frequent, ceased at about the ninth hour of the experiment. The animal made a complete recovery.

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*One box-tortoise after the injection, closed its shell and died thus, forty-five minutes after administration.

Experiment 41.—Common bat, received 0.005 *per orem;* within six minutes violent convulsions occurred, lasting three minutes, with two short and incomplete periods of relaxation, and terminated in death. Weight of animal 118 grammes, including skin and wings. The spasms were peculiar; such was the rapidity of the clonic movements that they could not be followed by the eye, and they resembled rather a tremor than a spasm. The same peculiarity is observed in the case of the mouse.

Here approximately the same dose proportionate to the weight of the animals, had been given in the case of the frog by the direct introduction into the parenchymatous fluids, in the mammal by the indirect path of the digestive tract, and yet the mammal died, the frog recovered. I have, in comparing the weight of different species experimented on, and accounting for individual idiosyncrasies, found the minimal lethal dose to be nearly the same in the different reptilian and amphibian classes.

The fallacy referred to has doubtless had its origin in the fact that if the frog be placed in water containing small amounts of strychnia, he will react very readily by convulsions, while other animals will not. The real reason for this vulnerability is to be sought for in the greater absorptive power of the frog's skin. Other aquatic animals (newts) having the same absorptive power have the clonic spasms ill-marked, but are nevertheless under the influence of the poison. Then again the advice is often given in hand-books on toxicology, to select only *small frogs* when desirous of testing for small quantities of the alkaloid, showing that the supposed vulnerability of the frog may have had its origin in the small size of the animals employed. Larger specimens in the same jar of strychninized water with smaller specimens, will frequently be unaffected, while the latter are in full tetanus, and a bull-frog* will even in a strong solution of strychnia remain unaffected for days, when the leopard frogs in the same fluid are dead, for the reason that the former has a thicker skin than the latter.

Another observation made was that if the extremities of an animal are placed in an unnatural position before the tetanus is established and maintained so, that the tetanic rigidity will preserve that position when the constraining apparatus is removed. For example, in the case of a bat the wings were secured by weights, one of the wings being folded, the other spread out. The weights were removed when the tetanus began, and although both wings were in combined clonic and tonic spasms, the extended wing remained extended, the folded wing remained flexed. The peculiar distortions noticed in some of the instances of chronic strychnia poisoning, were due to the confinement of the animals in narrow jars.

PROPOSITION II.

That strychnia requires, for its characteristic motor effects, a well developed central nervous system, and that therefore these effects are absent in the earlier embryos.

We have already seen that the higher the development of the nervous system, the more decided became the strychnia symptoms; since the embryo of higher animals presents us with an imperfect nervous system, let us see whether the dictum holds good here also.

Experiment 21.—The egg of a common barn-fowl of the sixth day of incubation was carefully opened with a graver, and the finely developed embryo exposed. Three drops of luke-warm egg-serum, containing 0.00015 strychnia, were slowly injected by a hypodermic syringe into the amniotic cavity. Previous to this the embryo had been moving around slowly. The heart was beating about 90 per minute; the auricles and ventricles filled well in their respective diastole: 22″, cardiac beats 70, well filled at diastole; 45′, animal quiescent, cardiac beats 42′, the interval is long, the diastole imperfect, the systole very abrupt, short and complete; 1′ 30″,

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*For a similar observation see the paper of Dr. J. J. Mason, on lead-poisoning in frogs. *Trans. Am. Neurol. Assoc.*, 1877.

diastole so incomplete that hardly enough blood enters the heart to give it color; there are from this time to 1′ 49″ only three short and hardly perceptible movements of the heart when death occurs.

Experiment 22.—The egg of a barn-fowl of the twelfth day of incubation was opened, as in the preceding experiment. In order to leave the embryo in as natural circumstances as possible, the solution of strychnia, containing 0.005 was merely dropped over the amniotic sac, and the gap in the shell closed by a cover glass. The cardiac movements cannot be observed in an embryo at this stage; and further than that the creature turned round on its axis several times, a movement common to healthy embryos at this stage; it manifested no symptoms, except that the movements became perceptibly weaker, and ceased in forty-seven minutes entirely. The embryo then became opaque, and was dead. The surfaces of the yolk and amnion were carefully washed, and the *liquor amnii* tested for strychnia, which, besides documenting its presence by a decided bitter taste, gave the characteristic reaction with bi-chromate of potash.

Experiment 37.—A large female rabbit was secured on the tray, and, being pregnant twelve days, the left corner of the uterus was exposed and laid over on the mesentery; one of the dilatations was then carefully opened by surface section, and the embryo, whose heart was beating 112 per minute, fully exposed. A drop of a strychnia solution, containing approximately 0.0025 of the acetate, was dropped on the amniotic opening. In 22″ the heart was beating 138; but at 1′ 15″ this had sunk to 85; the heart failed to fill at the diastole; some movements (vermicular) of the body occurred, which became gradually less frequent, and a commencing opacity of the tissues marked the death of the animal, the heart having ceased moving several times and then resumed action by a few imperfect contractions.

In none of the foregoing experiments could any hemorrhages be discovered in the area vasculosa, as marking an increased blood pressure. The only action of strychnia on the earlier embryos seems to be upon the heart, which organ, after an inconstant and brief period, during which the beats are increased in frequency, shows fewer pulsations than before. At the same time the diastole is imperfect. While an imperfect diastole in an adult animal or an advanced embryo would justify us in looking to the pneumogastric nerve as the factor, the undeveloped condition of the centres of that nerve in the medulla, during early embryonic life, forces us to seek for some other influence. Shenk, of Vienna, has shown that the embryonic heart when excised and maintained at the normal temperature in serum, will continue beating, as before, and that any portion of the organ, equivalent to one-twelfth of the whole, possesses the same power.

At this period of life the proper ganglia of the heart are not yet differentiated, and the cardiac motions must be referred to associated protoplasmic action, independent of nerve centres. Those protoplasmic powers which preside over the more passive function of the diastole are first *paralyzed* by strychnia, and then those presiding over the systole suffer. It is also observed that embryos whose circulation has ceased under the influence of strychnia, die much more rapidly than those whose circulation is arrested by excision of the heart, as marked by the early opacity of all the tissues. This shows strychnia to have a lethal influence on all protoplasm, when brought in contact therewith in sufficient quantities.

PROPOSITION III.

Excluding the influence of imperfectly oxygenized blood upon the circulation, the legitimate effect of strychnia on the circulation is to stimulate the systolic movements of the heart, to contract the arteries, increase the rapidity of the blood current and to raise the blood pressure.

The egg of a salmon, containing an embryo ready to escape from the egg-capsule, is placed in a watch-glass filled with water, and after the restlessness of the embryo has ceased, 0.0003 strychnia is added. Before this addition the cardiac pulsations varied between 18 and 24 per minute. 6″ after administration violent movements occurred, rupturing the capsule, and the animal escaped; in consequence of the excitement, doubtless, the pulsations rose to 26, but after half a minute, instead of returning to their previous rate, as under normal circumstances, they rose to 32. Systole short, quick and complete; the heart does not attain full diastole, the pectoral fins move more rapidly, so that the eye can hardly follow them.

A great advantage of this experiment lies in the complete transparency of the whole animal, and the distinctness and size of the blood corpuscles, so that the changes in the blood current can be readily traced under the microscope; in 39″, the capillaries in the tail became contracted, the movement of the aortic blood current, which previously required 6″ to pass the distance of one-fiftieth of an inch, now is so rapid as to require only one-third that time; and the same thing is observed in the segmental and branchial vessels.

1′ 22″, restlessness increases, the fish occasionally beats the water with the tail; in quiet intervals the bodily axis is seen to be curved, the cardiac movements shorter, less complete and 60 per minute, complete obliteration of terminal capillary arch of tail, movements of mouth, and operculum distinctly spasmodic; 1′ 45″, several of the veins of vitellus contain motionless blood corpuscles; the column gradually accumulates until a stasis of even the larger vitelline veins is produced; at 2′ 12″ the stasis has extended to the systemic veins, and the heart is now motionless, contracted and nearly empty; 2′ 30″, slight movements of the heart occasionally recur, although there is no circulation whatever, and the tissues of the animal are becoming opaque; the oral movements continue, and occasional motion of the body is noticed until 4′ 30″, when no further signs of life occur.

Experiment 310.—In the last experiment, an imperfect supply of oxygenated water, might be accused, although erroneously,* of having produced some of the symptoms described. To obviate a possible source of error, a larger specimen was placed in a specially arranged trough for microscopic observation, and by capillary tubing a constant stream of slightly strychninized water was kept continually running through the trough. The degree of concentration was such that the whole quantity of water in the trough at any one time was about .000000005, the entire quantity of strychnia to which the animal was subjected was 0.006. Before and during the first part of the experiment, the animal was very vivacious and it was difficult to keep a given part in field. Commencement, heart beats 36, oral movements rare, and come on only after excitement; pectoral fins in constant motion, circulation of vitelline vessels more active than in the last specimen, in capillaries of tail the rate is similar to that noticed in a frog's web, but without the local interruptions observed in the latter instance. No change occurs for 2′ 30″, when the animal becomes markedly restless and the cardiac movements rise to 64. At 5′ it is quiet again, the capillary and general circulation is at least twice as rapid as before, the capillaries are much more contracted than in a similar embryo under normal circumstances, at 7′ 45″. At 12′ the motion of the blood is occasionally checked ; on examining the heart at such times it is found arrested in tetanic systole. At 22′ this systolic contraction having recurred four or five times, the capillaries are found filled with stationary blood corpuscles, the arteries are contracted, the veins distended, and after a few spasmodic oral movements, death took place in 24′ from the commencement of the experiment, in tonic spasm.

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*Stasis occurs when a non-strychninized fish dies by asphyxia, in from fifteen to twenty minutes, but the heart is not found contracted.

Neither in these nor in similar experiments could any diapedesis of blood corpuscles be observed. At the same time the blood in the arteries was arterial *throughout.* This fact is readily determined in these almost glass-like transparent creatures, by the fact that in very thin layers their arterial blood has a clear yellowish tint, while the venous has a greenish hue, pronounced enough to enable us to distinguish between the two kinds. No movements were produced in the intestinal canal.

I have also confirmed the observation of Richter, that in the case of the frog's web and bat's wing, contraction of the arterioles and capillaries is one of the constant phenomena of strychnia poisoning, in this respect being similar to the fish's tail. I have further observed that division of the nerves going to the hind leg of the frog, in which instance the tetanus of the extremity is abolished, did not entirely abolish this contraction of the vessels. This has led me to infer that the increased arterial pressure is not exclusively due to the effect of strychnine on the central nervous system, and will have to be regarded distinctly from the effects of the alkaloid on the central nervous system.

I have not been able to derive any satisfactory results from the employment of the manometer and kymographion in mammals. The customary employment of chloral hydrate and curare, to paralyze the muscular contractions, adds countless sources of error to the experiment, and where we dispense with these antidotes, the elements of asphyxia, of muscular compression of the venous conduits, and the abnormal psychical condition, render any record by these delicate instruments futile. For the latter reasons also, the observation of the circulation in the pia, through a glass plate in the trephined cranial opening, could only be utilized in a general way. It seems that the arterioles contract just precedent to the first convulsion, but during this and all subsequent convulsions, as well as in a lesser degree during the intervals, venous engorgement is constant.

Our fourth proposition is already partly involved in the results of the foregoing experiments.

PROPOSITION IV.

That some of the effects of strychnia on the vaso-motor system are independent of its influence on the central nervous system.

Experiment 11.—Small *Rana fontinalis*, secured and web fixed in field of microscope, first under a Gundlach II, latterly under a Gundlach V. Strychnia 0.01 *per orem*, to avoid the undesirable effect of a hypodermic injection (it being known that with sensitive frogs a puncture of the skin will suffice to produce capillary contraction, or even momentary stasis). At the end of one minute the capillaries and arterioles gradually and generally contracted; at 1′ 50″, a tonic spasm, during which all the arterioles contract almost to obliteration, while the veins become fuller; 2′ 10″, several clonic spasms, during which the circulation becomes re-established; 2′ 45″, a second tonic spasm, the arterioles only in medium contraction, venous stasis for twelve seconds; 3′ 30″, arterioles considerably contracted, and this contraction increases during the next two minutes, while the animal is free from spasm; 8′, the nerves of the left posterior extremity are isolated and divided high up; during the progress of the operation the animal passes into tonic spasm, preceded by a series of clonic convulsions; after division the left hind leg was supple, while the rest of the frog was in tetanus; 12′ 30″, arterioles still contracted, vascular current accelerated, no peripheral layer of motionless corpuscles, nor diapedesis; 1 hr. 21′, arteries still contracted; during severe clonic spasms there is temporary and evanescent engorgement of veins; 1 hr. 50′, relaxation of spasm, arteries less contracted, but still not of normal calibre; 2 hrs. 2′, the arterioles become gradually contracted again, and with the almost total obliteration of some vascular areas, the frog passes into tetanus; observation closed.

Experiment 12.—In a young *Rana leopardina* the right hind leg is severed, the artery and deep vein alone being preserved; after the circulation in the web had returned to an approximately normal condition, 0.015 strychnia was injected into the limb; in 12′, complete and progressive contraction of the arterioles and capillaries; in 30′ tetanus.

Experiment 14.—In a small frog of the same species, secured in like manner, a similar division is made, but in addition the vein is divided; 0.015 strychnia injected into separated limb. The circulation in the web is much less interrupted under these circumstances, than one would suppose *a priori*, and a visible effect is produced by the alkaloid; in 19′, a notable contraction of the net-work; no tetanus.

Experiment 13.—Another frog of the same species, secured in like manner, has the spinal cord destroyed by passage of a sound, leaving the medulla and that part of the cord above the origin of the great brachial nerve intact. The vagi and sympathetic cords divided. Strychnia being administered (dose uncertain) the same contraction of the vessels in the web is observed. It seemed to be produced much more slowly than in an uninjured specimen, but the effect was marked and continued for five hours, no venous stasis occurred, and the contractions continued until death. Peripheral irritation (of the web) increased the contraction. Tetanus existed only in the head, and hyoid regions. Experiments 15, 16, 17, repetitions with same result.

Experiment 18.—Large *Rana leopardina*, cord destroyed as in Experiment 13; the animal previously received 0.05 subcutaneously. On placing it on the edge of the table, with the hind legs dependent, no œdema occurred in these parts. In non-strychninized frogs such œdema is almost a constant phenomenon under these circumstances.

From these experiments but one conclusion can be drawn: that the vaso-motor influence of strychnia is in part at least independent of the central nervous system, for the contraction of the vessels persists after the nerves are divided (Experiments 11, 12), and after destruction of the greater part of the cord (Experiment 15). The engorgement of the veins is also shown to be a mechanical phenomenon, for it reached its highest point while the animal was in tetanus, and was absent naturally when the vein was divided, as well as where there was no tetanus of the trunk muscles. The œdema following the vaso-motor paresis induced by destruction of the cord is therefore absent in strychnia poisoning. In this independency of strychnia with regard to its vaso-motor influence, it is analogous to nitrite of amyl, which, according to Huizinga, also may act locally on the blood-vessels.

PROPOSITION V.

That the peripheral nerves take no active part in the production of the typical strychnia symptoms, and that no anatomical changes occur in their structure.

Experiment 3.—The crural and sciatic nerves of a large toad were divided on one side in the altitude of the coxo-femoral joint, without loss of blood. The leg was completely paralyzed. Strychnia 0.1 injected under the skin; within 1′ full opisthotonos and clonic spasms of trunk, head, both anterior, and right hind leg. The divided member (left hind leg) remained quiescent throughout; the animal died in twenty-two minutes.

Experiment 10.—Large *Bufo*, 0.006 subcutaneously; usual prodromata, complete tetanus in 62″, during the progress of which, the nerves of the left hind leg were divided. The division was completed 6′ from the time the first tetanic spasm had manifested itself, and the limb which till now had been rigid and convulsed like the rest of the body, became as relaxed and quiescent as in Experiment 3, for four minutes. At the end of that time it participated slightly in a clonic spasm; on reopening the wound, I found a delicate branch of the plexus intact; on its division no further symptoms occurred in that extremity, the animal dying in four hours.

Sixteen other toads, in whom various nerves had been divided in the anterior and posterior extremities, exhibited correspondingly absence of tetanus in the muscles supplied by the divided nerves. The same was observed in mammals. I found in several of these instances that the discrepancy between authors with regard to the paralysis or non-paralysis of the motor nerves, can be easily explained. The reaction of the sciatic nerve was unaltered in those animals which, either on account of their size, or the smallness of the dose received, suffered only a moderately intense tetanus, and for a short time; but the longer the tetanus had lasted, the greater its intensity, the more the reaction became diminished. I had in the earlier experiments given large doses of the alkaloid to toads, and found them relaxed and apparently dead four or six hours later. In making autopsies of such animals, I found the heart still beating, and from this was led to watch other apparently dead toads for some further time before throwing them away, and was frequently surprised to find an animal previously supple, and of a cadaver-like softness, return to life (especially after a cool evening) in from six to twelve hours. In three instances I found a *repetition of the tetanus*, occurring on recovery; and with or without tetanus the recovery was never permanent, for the animals died in an asthenic state.

One toad was tested as to his reaction, while in the condition of apparent death, and this was found *nul;* when he again showed symptoms of animation, it was obtained.

In a large frog that had been in tetanus thirty-seven days, the nerves of the left hind leg were divided, the tetanus continued in the other leg as well as in the trunk, but the operated limb was quiescent, and on testing the exposed nerves of both sides four hours afterwards a difference of four centimetres found in favor of the left (operated) limb.

From these facts I am led to look upon the diminished reaction, not as the result of a specific paralysis produced by strychnia, but as analogous to the diminished reaction which follows *any over-exertion of the voluntary muscles.* That strychnia exercises no special influence on the nerves in this direction is shown by the fact, that where the nerves are divided, and where as the sequel will show, it can be chemically traced in the nerves of the operated limb, no difference from the reaction of the separated nerves of a non-strychninized animal can be observed, while the further the tetanus proceeds in the opposite member, the less pronounced the reaction.

Experiment 270.—Medium-sized *Rana fontinalis;* chronic tetanus had lasted seventeen days. The nerves of left hind leg divided as in Experiment 3; immediately the tetanus ceased in that limb, while it continued until the death of the animal (which took place two days later), in the rest of the body. The sciatic nerve of the unoperated limb was examined microscopically, and not the slightest indication of a change observed; its sheath was hyperæmic.

I have after numerous examinations made on the nerves of animals killed suddenly by large doses, as well as killed by the long-continued use of minimal doses, arrived at the conclusion, that strychnia produces neither primary nor secondary changes in the tissue of the peripheral nerves.

Having proved that division of the nerves instantly abolished strychnia tetanus, it behooves us to show whether the continuity of the nerves is sufficient to maintain the limb in tetanus, if the circulation is interfered with.

Experiment 38.—A large toad is secured on the operating table, and the integument of the right hind leg being divided by an incision encircling the limb, the crural and sciatic nerves are exposed and isolated. The blood-vessel accompanying the sciatic nerve is torn through purposely, and then the entire extremity is amputated, with the exception of the previously isolated nerves, which are kept moistened by serum. Hypodermic injection of strychnia 0.001. In 2′ violent clonic spasms of the whole body, *synchronous* spasms of leg and foot of amputated limb. The only difference between the right and the left leg was that the spasms of the femoral muscles were limited to contractions of the divided bellies, and produced no movements of the limb as a whole. The death of the animal occurred the next day, eighteen hours after operation; and while the tetanus continued in the body as a whole, to the time of death, it ceased four hours earlier in the amputated limb, its nerves having softened, and the tissues decomposed.

Experiments 39, 40, 44, and 42 confirmatory. These experiments signally prove that the muscular tetanizing influence is conveyed from the central nervous system to the muscles by way of the peripheral nerves, that it is not modified by the nerves, that the latter play merely the part of passive conducting mechanisms, and that as such, if the central nervous system is intact, they alone are concerned in conveying the tetanizing influence.

Experiment 43.—The left hind leg of a *Rana leopardina* is separated as in Experiment 38, the crural and sciatic nerves being left intact, the limb is then immersed in a saturated strychnia solution. Left therein for twelve hours; *no result.*

Experiment 47.—Repetition; same failure; the proximal extremity of the amputated limb was then permitted to touch the level of the fluid; in 16′ general tetanus; the mutilated limb which, suspended in a saturated solution for six hours, failed to show any spasms, now had them well marked.

Experiment 228.—Rabbit: left femoral and sciatic arteries ligated, extremity amputated, excepting the great nerve trunks, which were previously isolated (no chloroform employed); 0.006 injected into neck; animal in extreme tetanus in 18″; mutilated extremity agitated by clonic spasms, until death, 2 hrs. 20′. Care was taken to keep the limb warm, and the nerves moistened with serum. It was observed that there was a symmetry of the hind legs as to the time, rhythm and duration of the spasms. On testing for strychnia, no evidences were obtained from the distal part of the left sciatic nerve; faint traces were obtained from the right, and from the left above the line of amputation, which I did not consider very decisive; I therefore selected a larger animal and gave maximal doses of strychnia.

Experiment 229.—Large mongrel dog; 25 grains of strychnia acetas *per orem*,* 12 grains per rectum in solution. The left hind leg was then separated as in the last experiment, and as soon as the first symptoms of strychnia poisoning (labored respiration, horripilations,) showed themselves, which was 32′ after administration, one gramme more was injected in solution in three different parts of the body. During the injection the animal passed into a violent general tetanus which continued without remission 11′, when relaxation and death took place. The entire length of the sciatic and the greater part of the crural nerves of the right side were placed in one test tube, the same nerves of the left side below the line of amputation in another. Portions of other divisions of the nervous system were also preserved, the results of their examination will be referred to in another place. The contents of the first two test tubes, the one containing the left nerves designated as A, the other as B, were digested with a mixture of two parts of muriatic acid to ten of water for two days, until they were completely fluid, the emulsion was filtered, the part remaining on the filter being redigested and refiltered, and treated with commercial alcohol as long as anything more would dissolve. The residue was repeatedly washed with water and filtered. The resulting fluid was evaporated to dryness in a water bath, and this residue in turn treated with alcohol, for three days, filtered and evaporated, again dissolved in water and again filtered. Now liquor ammoniæ was added in excess, and the whole shaken up in a flask with chloroform. The latter having subsided was then drawn off with a pipette, and rapidly evaporated on a water bath; the dry residue then moistened with sulphuric acid (monohydrous) and exposed to the water bath again. The charred matter was finally dissolved in water, again treated with an excess of ammonia and chloroform, this procedure being repeated thrice, until the sulphuric acid ceased to char the evaporated residue, when it was suspended in chloroform; a drop of this solution after partial condensation by evaporation was dropped on the bottom of a warm porcelain capsule; as each drop evaporated another was added. As soon as the capsule was cooled off, the residual coating was moistened with strong sulphuric acid, a minute crystal of bichromate of potash being laid in the centre. In specimen A no result followed, in B the spot turned successively blue, violet, a beautiful crimson, red and green; the latter color remaining permanent.

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* Wrapped up in meat.

Thus the presence of strychnia in the muscles and nerves themselves is shown to be unessential to the production of tetanus.

PROPOSITION VI.

That strychnia increases the sensibility of the peripheral organs of sense, but that it is an error to therefore designate the convulsions of strychnia as the sole result of an increased reflex excitability of the cord.

Regarding the first part of the proposition there are no differences of opinion among observers. Strychnia in therapeutical doses has a marked effect upon the peripheral sense apparatus, which effect is even more patent in pathological cases. We see this very clearly in the case of amaurosis, especially when due to toxic influences (nicotin poisoning); a recent writer has indeed found that by brushing a solution of strychnia on the regio olfactoria of healthy as well as of anosmic individuals, the acuteness of the sense of smell could be increased. If this observer's (Möller) results are confirmed* they clearly prove that the heightened sensibility is in part at least peripheral, whatever share the heightened functional activity of the nervous centres may have.

If the old dictum, that the strychnia tetanus is solely adducible to an increased reflex excitability were correct, then the occurrence and intensity of the spasms would constantly depend upon the occurrence and intensity of the peripheral irritation. That is far from being the case. One frequently observes a frog passing into tetanus, on irritation, who recovers, and crawls away or sits still, in spite of frequent repetitions of that irritation. When left to himself, and while everything is quiet, he suddenly develops an exquisite tetanus. In a large goat, poisoned with a large dose of crystalline strychnia, and where on account of the slight solubility of the alkaloid in the gastric juice, the symptoms were delayed a long time, it was observed that the prodromal horripilations came on without any regard to the occurrence of disturbance. But this question can be submitted to crucial test.

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* I confess that it is very difficult to control such a difficult operation as painting the regio olfactoria with such a solution, properly.

Experiment 282.—Large bull-frog; the brain and spinal cord exposed for their entire length; the posterior roots of all the spinal nerves, the auditory trigeminus (which, in the frog, includes the glosso-pharyngeal) and optic nerves divided. All sensory impressions, except those of smell and the visual sensations were, therefore, excluded. To remove the former, the olfactory lobe was separated from the cerebrum. Fifteen minutes after the operation, 0.0015 strychnia injected into dorsal wound. Within one minute complete opisthotonos, preceded by a brief clonic spasm. The tetanus presented the same picture as that of an uninjured frog, except that jarring the table failed to bring on the spasm, although direct irritation of the cord and medulla did so. The spasms, however, became more anenergetic towards the end than in an unmutilated frog.

When the animal was picked up and thrown down violently, it responded like an uninjured frog, by a tetanic spasm; but here, probably, the direct influence upon the nervous centres by concussion was the causal factor: for the skin could be pinched, the toes cut away singly, the leg amputated, and the eye pinched, without any such result.

Experiment 296.—Kitten twelve days old; division of posterior roots of all the dorsal and cervical nerves; then division of cord at atlo-occipital articulation, and also below middle dorsal region. Where the roots were left undivided and a corresponding division of the cord was made in another kitten of the same litter, it had been previously ascertained that perfect tetanus ensued in the head, trunk and anterior extremities when strychnia was administered. In the present instance 0.01 strychnia injected into dorsal wound; in 40″ trismus and extension of head; at 3′ 45″, a sudden tetanus of the trunk and anterior extremities, passing into clonic spasms, with which the animal died at 6′.

Here all sensory influences on that segment of the cord from which the nerves of the trunk and anterior extremity take their origin, were abolished, and yet we obtained the typical strychnia spasms. The delay in the response of the cord noticed in the last experiment will be discussed elsewhere. The experiments of Freusberg, Mayer and Möller have paved the way for regarding strychnia as a primary irritant of the grey matter, a view which my experiments fully confirm.

That peripheral irritation increases and maintains the tetanus and spasms, remains, nevertheless, a fact; and this has been recorded by the earliest experimenters. What, therefore, the originality in the recent suggestions of a French writer consists in — who recommends darkness and quiet as important adjuvants in the treatment of strychnia poisoning — it is difficult to see. The older physiologists have long ago noticed that recovery is more likely, under these circumstances, and almost every therapeutical hand-book embodies these suggestions. I have employed darkness and quiet as most important aids in keeping frogs alive for weeks, under the influence of the alkaloid. [See chronic strychnia poisoning.]

PROPOSITION VII.

That strychnia acts upon the entire grey matter of the central nervous system; that the characteristic tetanus is due to the effect on the axial tract (central tubular grey matter), whose upper extremity (pons, medulla and cervical cord) is more concerned in the production of the spasms than the lower (lower cervical, dorsal and lumbar cord).

The question as to whether strychnia is to be considered as exercising a *cerebral* or a *spinal* tetanizing influence, is the most disputed one of this subject under discussion at the present time. Two of the most recent experimenters flatly contradict each other on this point — Mayer claiming that the spinal cord *per se* could not produce spasms, under the influence of strychnia; Freusberg, on the other hand, asserting that it is the part chiefly concerned. We shall find that both are wrong, and that the truth lies between.

Experiment 50.—Large toad; lumbar cord divided crudely as only the left hind leg hung passively, it was to be presumed, and the autopsy confirmed this, that only a part of the cord and *cauda* was divided. Gave 0.005 with Pravaz's syringe into peritoneal cavity; in 1′ accelerated flank movement, in 4′ clonic and tonic spasms of whole body, except left hind leg.

Experiment 54.—Toad; division of cord higher up, above origin of crural nerves; 0.005 strychnia; perfect tetanus of head, trunk and anterior extremities; posterior extremities relaxed, and quiescent until death.

Experiment 55.—Toad; division of cord, a millimetre below lowest brachial nerve; absence of tetanus in hind legs; tetanus of trunk less perfect than in Experiment 54.

Experiment 56.—Large *Rana leopardina;* cord divided immediately above the point where the greater brachial trunk is given off; the animal was allowed twenty-four hours to recover. As frequently has been observed, several hours after operation, *spontaneous* tetanus (rigidity) occurred in the hind and fore legs, from which the animal recovered. Twenty-four hours after operation, 0.005 strychnia subcutaneously. 3,′ pupils dilated, eyes protrude, tetanus in head (hyoid, extensor capitis, ocular and mandibular muscles), with "lifting" synchronous movements of fore legs. During this period of perfect head and imperfect fore leg tetanus, the movements described by Pflueger would occur, as in non-strychninized frogs, when the board on which the animal sat was tilted; that is, the hind legs would recover themselves when there was danger of falling. 2 hrs. 40′ later, a second dose of strychnia, 0.0025, was given; this was added to the water in the jar, to which the animal was transferred. The following morning the head and fore legs were still in the same tetanus as the day before; in the course of the day, occasionally, similar spasms could be provoked in the hind legs on irritation; these were not synchronous with the head spasm, and consisted of an imperfect extensor spasm, produced by violent irritation, such as throwing a book on the table. During the afternoon, the head tetanus continued unabated — frog still in strychninized water — but no spasms whatever in posterior segment of body; the fore leg tetanus became weaker and weaker, and was finally abolished. The third day, the head spasm was very marked, no other part of the body affected. In the afternoon, animal apparently dead; on opening body, heart found beating, regular and full, at the rate of eighteen beats per minute.

The disappearance of the fore leg spasm after it had once become established, points to a disorganization of the lowest part of the anterior segment of the cord, produced by the operation and resulting hemorrhage; it has, therefore, no essential bearing.

Experiment 160.—Large toad; division through anterior part of optic lobes; instant ataxic paresis of all members; occasionally, chorea-like movements. Injection of 0.005 strychnia subcutaneously; tetanus perfect, as in uninjured animals, with the sole difference that reflex tetanus would not occur on exposing the animal to a bright light.

Experiment 117.—Toad; division just below cerebellar valvule; after operation, tonic flexion of trunk and extremities, these being folded inwards. On injecting 0.005 strychnia, violent reflex struggles to free the extremity injected. When this animal jumped, previous to being put under the toxic influence, it regularly rolled over on its back; during the inception of the tetanus it rolled over in like manner. Tetanus general but not typical; in one hour and twenty minutes, death.

Experiment 114.—Incomplete destruction of medulla; usual strychnia tetanus.

Experiment 115.—Toad. Complete separation of medulla from cord; a second division below cerebellum; tongue protruded during operation; apparent death. Tetanus as usual, except that the tongue remained protruded.

Experiment 116.—Two transverse incisions in nervous axis of toad, the anterior separating the cerebellum from the optic lobes; the posterior one-eighth of a millimetre below calamus scriptorius. There was complete retraction of left eyeball, and contraction of left pupil; protrusion of right eye and dilatation of right pupil, as a result of operation. Strychnia 0.005 subcutaneously; tetanus of tympanic, hyoid and cephalic muscles within 2′, followed in 4′ by general tetanus of trunk and extremities. At no time were there any spasms of ocular muscles; these remained as before administration.

Experiment 124.—Division just above first cervical pair in toad. Strychnia 0.0025; well-marked tetanus of head and upper part of trunk, none of body or extremities.

Experiment 201.—Same division in a *Rana leopardina.* Tetanus general.

Experiment 202.—Same division in another *Rana leopardina.* Strychnia 0.0025; well-marked tetanus of head within 1′; for eight minutes the members were perfectly quiescent; at the end of that time the extremities shot out in ideal tetanus, and remained so for twenty-eight minutes, when relaxation of these parts took place, although the head was still rigidly tetanized. During the following half hour, the head spasms became clonic, the shocks becoming gradually more marked and frequent; and at the end of this period, the extremities again shot out, and were agitated by clonic spasms. When these ceased it was easy to renew them by appropriate irritation. From this time on, the spasms of the extremities became less perfect and rarer, while those of the head continued unabated until death—four hours from the time of administration.

Experiment 203.—Same operation in same species. The animal was frequently pinched, thrown on the floor and otherwise disturbed after the poisonous agent had been administered. The extremities showed no tetanus until seven minutes from the time the head had been affected.

Experiment 204.—*Nannemys guttata* (small yellow spotted turtle). Division of cord between roots of second and third cervical nerves; strychnia 0.01; tetanus of head and hyoid muscles, none of retractor capitis or extremities.

Experiment 205.—*Chrysemys picta* (painted turtle). Division through calamus scriptorius; strychnia 0.015; complete and general tetanus.

Experiment 319.—*Rana leopardina*, which had been in strychnia tetanus for eighteen days. Cord divided through region of first brachial nerve; instant cessation of tetanus in extremities, but continuance of tremors* in same.

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* These tremors are not essentially strychnia symptoms. See sequel.

The evidence of these experiments is, on first sight, somewhat confusing. This much is certain, however, that the lower down the cord is divided, the more certain are we of rendering it impossible for strychnia to tetanize the muscles deriving their nerves from the lower segment. Division in batrachians of the dorsal portion of the cord, will prevent tetanus in the hind legs (Experiments 50, 54, 55); in the majority of animals division, as in Experiment 56, will check tetanus in all extremities. It further appears that division above the origin of the brachial nerves, even when it does not prevent tetanus, will delay the spasms for an appreciable period of time, varying from three to fifteen minutes (Experiments 201, 202, 203). The delay is manifested by the relatively earlier occurrence of the head tetanus; and that it is not due to lack of reflex irritation, a comparison of experiments 202 and 203 will demonstrate.

Division above or in the latitude of the calamus scriptorius, exercises no influence on the general spasms.

Altogether, these experiments show that the further removed any portion of the cord is from the medulla oblongata, the less likely is it to react on strychnia by itself, and that in regard to strychnia, as in some other respects, the medulla deserves the name of a veritable convulsive centre.

All animals are not alike in this respect. In tritons, tadpoles, salamanders and fish, section of the dorsal cord will not check tetanus in the muscles supplied by the part behind the line of section. It is singular that a similar result should be obtained in the case of the newly-born of higher animals.

Experiments 146–150, inclusive.—Five kittens of one litter, thirty-six hours old, were each injected hypodermically with 0.05 strychnia. In three, the section of the cord was made before convulsions appeared; in two, after.

Experiment 146.—Section through lower cervical cord, before convulsions had appeared. Subsequent tetanus general; pupils *contracted;* death in 6′.

Experiment 147.—Section through lower dorsal cord before tetanus. Subsequent tetanus well-marked, except in posterior extremities, where it was feeble; but the tail was in the typical erection.

Experiment 148.—Section one millimetre below obex. Subsequent tetanus general; pupils dilated.

Experiment 149.—Section through middle dorsal cord; after tetanus became established, no relaxation of posterior extremities; death in 12′.

Experiment 150.—Section through lower dorsal portion of cord, after established tetanus. Tremors of hind legs follow the section, which gradually abate, leaving these parts in rigid tetanus; this in turn passes away, but is brought on again by pinching the tail. This again abates, and death takes place in 19′.

Here, while section of the cervical and upper dorsal cord did not have the effect of checking the spasms, which effect was noticed in adult amphibians and reptiles, and which we shall also find in adult mammals, still we find that division in the lower dorsal region diminished, if it did not check the tetanus in the hind legs, thus confirming the truth of our general proposition.

In older animals the effect of such section is much more pronounced.

Experiments 172–176, inclusive.—Five kittens of the same litter, twelve days old, treated as the last set, three being strychninized after, two being strychninized before section of cord, in various attitudes. Of the former group, one (Experiment 174, corresponding to 148 of last series) died before any observations were made.

Experiment 172.—Division of lower cervical cord before tetanus. Subsequent tetanus general, but trismus preceded the general tetanus by 1′ 15″; death in 3′.

Experiment 173.—Division of lower dorsal portion of cord, before administration. On administration, no tetanus of posterior segment of body; at 5′ 30″ a twitch of the tail occurred, which was of momentary duration, and was not repeated; death in 29′.

Experiment 175.—Division of middle dorsal cord, during opisthotonos. At the moment of division a fibrillary tremor occurred in the hind legs, and the tail became relaxed. The whole animal then passed out of spasm; but within 45″ it became convulsed in head, fore feet, and trunk; 20″ later this extended to posterior segment of body. On irritating the posterior extremities, the spasms became intensified. Death in 18′.

Experiment 176.—Section of junction of dorsal with lumbar cord, in altitude of last dorsal nerve, during opisthotonos. Cessation of tetanus in hind legs and tail; death in 20′.

Experiment 177.—Half-grown pup. Division of cord between atlas and axis: artificial respiration; strychnia 0.05, subcutaneously. Tetanus of head-muscles; trismus in 1′ 30″; in 4′ an imperfect clonic agitation of body, which ceased after having lasted five seconds, and recurred, but with still more diminished intensity, three minutes later. During all this time, the trismus and oculo-motor spasms were extreme. At the time of death only the head was affected (12′).

Experiment 152.—Full-grown terrier. Chloroformed; cord divided at seventh cervical vertebra. Before recovering from anæsthetic, 0.075 was injected under skin, and within one minute the head was in tetanus, and the fore legs were in a fibrillary spasm, which progressively merged into clonic convulsions. Death in eighteen minutes, no tonic or clonic spasms having occurred in posterior extremities.

Experiment 153.—Full-grown mongrel cur. Cord divided between second and third cervical vertebra. The operation was performed hastily, to give the animal the best chances of recovery, and was permitted to recover from the effects of the chloroform. Strychnia 0.06, subcutaneously. In 1′ head affected with the most extreme tetanus, the teeth grating on each other. Artificial respiration was maintained; and the pupils were observed to be moderately contracted. For nine minutes not the slightest movement occurred in the trunk and extremities, although peripheral irritation was not wanting (respiratory manipulations). Suddenly, and without premonitory symptoms, a violent tetanic spasm agitated the extremities, trunk and tail, lasting 2′ 30″, and giving way to complete relaxation; during this period of relaxation, the head continued in spasm, the jaws being firmly set, the ears and nostrils twitching, the extensors of the head being in contraction. *As long as artificial respiration could be maintained, the pupils were contracted;* as soon as it was suspended, they became dilated. *During the suspension of the artificial respiration, no spasms occurred in the trunk, nor were those of the head intensified.* After a quiet interval of five minutes, the tetanic spasms became permanently established, and were synchronous with those of the head. Death, 36′ after administration.

On the autopsy (given fully under the head of "pathology") it was found that the cord had not been completely divided. The anterior fissure of the cord had not been reached, and part of the right anterior and the whole left anterior, with part of the left lateral columns, were intact. Therefore the passage of the tetanizing influence of the medulla oblongata, although at first impeded, was, when once established, permanent. As a collateral result we observe that when the disturbing element of asphyxia is removed, that contraction and not dilatation of the pupil results.

Experiment 210.—Half grown dog, cord divided with the exception of the anterior columns, in the same altitude as in the preceding experiment. Interval between first head and first trunk spasm (which came on as abruptly as in 210), a little over four minutes. General persistent tetanus after twelve minutes; completed division of the cord at 14′, whereupon the spasms continued in the head as heretofore, those in the body instantly ceased; at 17′ a weak spasm of a clonic character recurred, and ceased in ten seconds, but the head spasm continued until death: 18′.

Experiment 213.—Terrier; cord divided as incompletely as in 210 and 153; interval between first head and first trunk spasm, 6′. Completion of section at 9′, cessation of trunk spasms as before. Thirty-three minutes later, clonic spasms lasting 11′, began in the hind limbs spontaneously, gradually involving the trunk and fore legs, until the trunk was in complete opisthotonos; relaxation followed lasting 2′; second tetanus (since completed division) partially clonic, lasted 3′ 30″, interval 6′; third tetanus, less energetic, interval 11′; fourth spasm momentary; no further spasms of trunk or extremities until death (2 hrs. 10′ after administration). During all this time head tetanus continued with *undiminished* intensity. Apparent death 2 hrs. 5′, after administration; on opening thorax, heart found beating slowly, imperfectly and infrequently.

Experiment 214.—Terrier; complete section of cord in same place, no chloroform used, strychnia 0.10 hypodermically; first head spasm 3′ after pupils contracted, first body spasm 22′ later, gradually weakening in energy as in 213. Death in 1 hr. 15′, head spasms energetic to the end. Rigor mortis came on immediately after death in head, not established in trunk and limbs for three hours.

These experiments show that even high division of the cord will delay the occurrence of strychnia tetanus in the trunk and extremities for from ten to thirty minutes, especially if the dose has been moderate. It is also noteworthy that the tetanus of the cord is less energetic, and ceases sooner, than that of the medulla oblongata. It appears that the lower down the cord is divided, the earlier is its "strychnia excitability" exhausted. That the maintenance of long-continued convulsions depends upon the intactness of the communications with the medulla oblongata, is proven by Experiments 210 and 213, where, while there was some delay of trunk spasms after partial division, these spasms were perfect and energetic when once established, but when the division was completed, these lost in intensity and frequency. This seems to prove that a certain accumulation of irritations in the medulla is necessary before the tetanizing influence can pass the narrow bridge of white substance left in the former instance, and that when this bridge is destroyed, the removal of these influences is manifested in the weaker character of the spasms of the completely separated lower cord-segment. It also points to the anterior columns, as the conductors (in part, at least) of the tetanizing influence. Several experiments which I will not take the space to record, prove that division of the posterior white columns and the posterior grey cornua, fails to check the convulsions in the slightest; to some extent this strengthens the view that increased reflex irritability is not as important a factor in the production of strychnia tetanus as is often supposed.

I do not think that there is any source of error in these experiments: that, for example, the division of the cord itself, or of its vessels, interfered with the legitimate effects of strychnia. If a suspension of the blood supply were the cause of the suspension of the tetanus, why should the latter be most marked in case the lumbar cord is divided, and least marked where the upper cervical cord is cut? In the latter instance the blood supply is interfered with most; in the former, on account of the numerous branches from the lumbar and intercostal arteries, not at all (practically). As wrong would it be to state that spinal shock had delayed the usual strychnia manifestations: for in several experiments we have permitted the animal to recover from this shock, and yet had the same result. Then in the beautiful and crucial experiment, where the cord of a frog in chronic strychnia tetanus was divided, we had instantaneous cessation of the tetanus, but a continuance of the tremors, due to an artificially induced pathological condition, thus clearly and amply demonstrating that the shock of a section does not affect the convulsibility of the cord *per se*. I feel rather inclined to seek for a source of error in the inferences of those, who not only do not content themselves with stating that the strychnia tetanus is essentially spinal, but go so far as to claim that division of the cord increases its intensity. It is true that occasionally a convulsion occurs after division, which is, if not more violent, at least as violent as the severest strychnia spasm, being both maximally clonic and tonic combined; but this very spasm occurs in many animals, who, without receiving any strychnia, have the cord divided; it is not a strychnia convulsion at all!

Our general conclusion with regard to warm-blooded animals is that strychnia affects the entire tract of grey matter, but that, in adults, as a rule, its influence on the cortex and on smaller segments of the cord is unnoticeable, if given in moderately poisonous doses. That the older the animal, the more the strychnia-excitability of the cord is diminished, until, in the adult, it may become almost zero in the dorso-lumbar region. The maximum of excitability is confined to a region bounded by the anterior border of the pons in front and the level of the second cervical nerve root behind. In this respect, there is no noticeable difference between the mammalia and the acaudate batrachia. When very large doses are given, premonitory spasms (monospasm, Experiment 1) are sometimes observed; these may be cortical, the horripilations and panniculus spasms may be referred to the hemispheres likewise, since they probably have a psychical motive.

PROPOSITION VIII.

That convulsions, although characteristic of, are not essential factors of, strychnia poisoning; and that strychnia can produce death, without the complications of nervous exhaustion, asphyxia, or cardiac paralysis.

I have already registered several experiments in which death occurred so rapidly in mammals, that there was not time enough for the development of asphyxia, as in the case of the bat, an animal which will survive many minutes after ligation of the trachea, and dies in a few seconds under the influence of strychnia. We have also seen that in lower forms of life, spermatozoa, leucocytes, infusoria, *et cetera*, death occurred without any vital manifestations. It remains for us to see how far this holds good for the mammalia.

Experiment 80.—Half-grown cat; jugular vein exposed, secured above and below an incision, through which 0.4 strychniæ murias is injected. Ligatures rapidly tightened, animal loosened, jumps over and falls on its back, in a violent momentary opisthotonos; death in 47″ from time of injection. The pupils were in medium contraction, the tongue caught between the teeth, heart beating feebly and tremulously for one minute and a half longer.

Experiment 81.—Puppy; 0.2 strychniæ murias injected into external jugular vein; almost instantaneous twitches of entire body; apparent death in 1′ 5″; thorax opened; heart still beating; stoppage of both auricles and ventricles in systole, at 3′ 15″.

Experiment 83.—Large cat; secured; 0.6 strychniæ acetas rapidly injected into thyroid branch of common carotid; instantaneous struggle (not tetanic), and death; autopsy: heart in rigid systole; brain moderately injected; no venous engorgement.

Experiment 96.—Large cat. Chloroformed; 0.5 injected, as before (canula pushed forward to lumen of carotid trunk). Death instantaneous; brain anæmic.

As an appendix to this chapter I will add one experiment, which sustains the inference that the legitimate effect of strychnia is contraction and not dilatation of the pupil. In order to test this properly, it is necessary to eliminate the disturbing influence of the asphyxia, which was already done in Experiment 153, by maintaining artificial respiration. In that case the reader will recollect that the pupils were contracted in the free intervals. This factor can be more directly eliminated.

Experiment 193.—Full-grown rabbit. Division of both cervical sympathetic nerves; medium contraction of pupils as a result. 0.05 subcutaneously; first spasm at 1′10″; with the spasms the pupil became progressively more contracted, and at length reached a pin-hole size; it returned to its previous condition just before death, which occurred in 40′.

That the irritability of the retina might not be accused of having caused this contraction, I repeated this experiment in another rabbit (Experiment 307) whose optic nerves I had divided the day previous; the same result was observed as in Experiment 193.

IX. THE PATHOLOGY OF ACUTE STRYCHNIA POISONING.

The first question presenting itself in relation to this topic is, whether any accurate micro-chemical test for strychnia can be applied to the nervous tissues. My search for such a test was in vain; but I have accidentally discovered an appearance, which, although not sufficiently decided to admit of any mature conclusions being derived therefrom, may lead to further inquiry. In the cord of a large cat which had been poisoned by a large quantity of the alkaloid (Experiment 1), after immersion in pure bichromate of potash solution (1:100 water) a peculiar greenish tinge was observed in the pia and the grey substance of the cord. As no alcohol or glycerine had been brought in contact with the specimens, and as the bichromate is not reduced by undecomposed animal tissues, I am at a loss to account for this appearance on any other basis than a strychnia reaction. The greater quantity of strychnia in the vascular pia and grey substance would account for the greater intensity of the color here; in fact, the white substance proper, aside from the processes of the pia and the grey cornua, did not present the tint in question at all.

Microscopically, this green color was equally distributed in the cells, septa and neuroglia.

I have also found it impossible to test for the minimal quantities of the poison which are found in the various organs, *quantitatively*. I can only infer that the grey substance contains more than the white, from the fact that those portions of the nervous centres containing most grey substance gave the clearest reactions; thus the medulla oblongata shows it better than the cord. That this preponderance in the grey substance is not due to a special affinity, but to its greater vascularity, I have hinted above. Gay claims that there is more strychnia in the medulla oblongata than in the cord; and the fact that I have often found it here, when examination of the cord gave a negative result, speaks in his favor.

Qualitatively, I have found strychnia, when administered in large doses, in the olfactory lobes, retina, cerebro-spinal fluid, basal ganglia, cortex, crura, cerebellum, medulla, cord and peripheral nerves. In short, it is probably universally distributed through the nervous system.

Neither in freshly-prepared nor in hardened specimens could I discover the slightest anatomical change. The nerve fibres, the ganglion cells, the neuroglia and the white substance were ideally healthy. I am aware that others have described changes in the contours of the cells, destruction of the nuclei, and molecular alterations of the protoplasm, but there is not one of these appearances that cannot be referred either to imperfect observation or improper manipulation. I have employed sections of cord, obtained from animals dying in extreme tetanus, to demonstrate the normal anatomy of the parts. The softening noticed by other writers is either cadaveric or collateral to hemorrhages, or due to serous exudation from the over-distended blood-vessels, when occurring in cases of acute duration.

The vessels are uniformly stated by various writers to be engorged. Most of them correctly ascribe this engorgement to the asphyxia and muscular over-exertion, not to a local effect of the alkaloid. It therefore becomes a secondary result — not an essential feature of strychnia poisoning. This is borne out by the fact that animals dying suddenly with either no, or very few, convulsions, have a rather bloodless brain, while the longer the convulsions have lasted, the more intense does the venous engorgement of the nervous centres become. As an illustration, I append the autopsy record of some of the more suggestive experiments in this essay.

Autopsy of No. 153.—Heart half relaxed, lungs slightly congested, prevalently on the right side, on which the animal had lain during the experiment. Abdominal viscera of normal appearance; large veins filled with lake-colored blood; arachnoid fluid clear; surface of cord sections injected; sinuses moderately filled with lake-colored blood; cerebral veins of both convexity and base, spinal and vertebral veins, moderately filled; arteries of larger calibre empty; arterioles well filled; no capillary injection of brain. Microscopical examination of cord and medulla, as well as of superior cervical ganglion failed to show any change.

Contrast this with the ordinary result, as obtained from an unrecorded experiment: Dog. 0.06 subcutaneously; in typical convulsions for two hours, with intervals; not observed after that period, and found dead eight hours after administration. Pupils in maximal dilatation, rigor mortis, lungs collapsed, ecchymotic, pleura costalis ecchymotic, ventricles of heart empty, left auricle same, right auricle and great veins filled to distention with dark-colored blood; liver, spleen and kidneys congested and soft; the blood runs profusely from the cut surface of these organs; bladder contracted and empty.

Arachnoid fluid tinged with blood; pia of cord, base of brain, and convexity intensely injected; veins and sinuses engorged. Hemorrhages had taken place at various points, chiefly sub-pial, but in one locality into the arachnoid space. One large coagulum had been poured out around, and had torn the radicles of the fourth cervical nerve; the grooves between the pons and trapezium, between the latter and the medulla proper, as well as the posterior perforated space, were obliterated by hemorrhages.

This picture is so similar to that found after asphyxia from strangulation, that its cause instantly suggests itself. It is not due to the strychnia primarily any more than the same venous engorgement found in tetanus, hydrophobia and epilepsy is due to the respective primary influences. That the heightened blood pressure in the arteries and the accelerated blood current may increase an already established engorgement — and thus the latter become transferred to the intermediate capillaries as a capillary stasis, produced during the final agony — does not militate against this view.

To obtain a perfect idea of the mode by which the venous engorgement is produced, it is desirable to observe its production in the living animal.

Experiment 185.—A half-grown dog is secured on the tray, and the obturator membrane divided so that the two longitudinal veins on its visceral side are not injured. This is accomplished by a crucial incision and cutting away the flaps piecemeal with a curved scissors. On now flexing the dog's head, the upper part of the cervical cord and the lower of the medulla oblongata become visible as far as the nodulus cerebelli. The surface of the columns is white, with delicate vascular rami; pulsatory excursions of arachnoid fluid invisible; respiratory excursions marked. The ventricular choroid plexus is rosy in hue, as also the ventricular floor and the cerebellar surface, though much lighter. Strychniæ acetas 0.015 was then injected under the nuchal skin. 22″, pallor of cerebellar cortex, vascularization of cord less patent; at 45″ this increases; the respiratory oscillations of arachnoid were marked. The choroid plexus now becomes redder, in contrast with the cerebellum, and a convulsion interrupts the observation momentarily. The head is forcibly flexed, but a stream of venous blood running from the cranial cavity obscures the view. As soon as this coagulates it is removed, and the apex of the ventricular floor is found injected, the choroid plexus purplish, and large vessels, previously invisible, are now seen in the pia mater. The purplish injection gradually fades during the next four minutes, but recurs during the next spasm, reaching a more intense degree than previously. This was repeated twice; on the last occasion there was another escape of venous blood between the cerebellum and medulla, during which the animal died. As long as the arteries could be observed, it was evident that they did not return to their normal calibre in the quiet interval.

Experiment 71.—Cat. Trephined and dura excised; glass plate inserted; received 0.06 the following day. Tetanus, as usual; no arterial engorgement observed prior to same, but more marked with each spasm, reaching its highest point in the terminal convulsion. Microscopical examination of medulla oblongata and occipital cortex: nerve elements healthy; capillaries filled with blood discs, whose outlines are distinct; agglutination in rolls frequent. Red corpuscles crowded in the adventitial and perivascular spaces; an increase of the round (lymphoid) bodies in the neuroglia.

I did not in my previous contradiction of the asserted pathological findings of recent writers take cognizance of the lesions which have been described as occurring with traumatic tetanus and hydrophobia, two conditions presenting many points of analogy to strychnia intoxication. Of the lesions described by Meynert in connection with the former affection, I shall have occasion to speak later. As to hydrophobia, Benedict has described "peculiar fatty or granular bodies" in that affection in Virchow's *Archives;* his figures clearly show the artificial nature of these bodies.

That the condition of the blood, as to quantity and quality, can have no influence in determining the tetanus, could have been inferred from the experiments of Salkowski, who produced tetanus as readily in frogs deprived of their blood, and having that fluid replaced by a saline solution as in normal animals. I have verified the latter experiments; an elegant variation of Salkowski's experiments consists in keeping the saline supply a constant one through a capillary glass tube, permitting the wound in the lower end of the divided ventral vein to remain open. The strychnia may be injected hypodermically, or better still, added to the saline solution.

A final dilatation of the arterioles and some capillaries can be often observed to take place just previous to death in the frog's web, the rabbit's mesentery, and pia mater. It is probably like the anenergy of the muscular system correspondingly observed, a symptom and result of over-excitation.

I refrain from appending a series of observations on frogs and toads confirming the conclusions just obtained, and would merely mention, that a deep injection and occasionally a hemorrhage under the velum of the fourth ventricle were noticed in every instance where the tetanus had lasted a long time. On microscopic sections of the medulla and cord of a turtle, I found numerous capillary hemorrhages, without alteration of tissue; the maximum of the change was in the spinal cord above the roots of the second cervical pair, and in the lower half of the medulla oblongata. The same was noticed in other animals, whether they had died of strychnia poisoning, or ordinary asphyxia; it has also been found in those dying in the status epilepticus. The localization of the hemorrhage is therefore doubtless due to mechanical factors.

X. THE PHYSIOLOGY AND PATHOLOGY OF CHRONIC STRYCHNIA INTOXICATION.

I have not been able to find any statements in the literature with regard to the experimental induction of chronic strychnia poisoning.* Schroeder Van der Kolk reports the example of a dog who was under strychnia four days, and finally recovered; and other observers have casually mentioned that frogs often remain in tetanus for several days, but none have, to my knowledge, made any systematic observations in this direction.

After numerous failures with the hypodermic method, I adopted the following procedure: A series of sixty-two specimens of *Rana leopardina* were placed in as many numbered glass jars. A record was placed with each jar, and enough water being allowed each frog to moisten the under surface of his body, a drop, or several drops (according to the size of the animal), was added from a standard solution of strychniæ acetas, containing 0.001 to the drop of water. The experiments were performed in the winter of 1877–78, in a permanently dark chamber, only occasionally illuminated by a candle, when the records were revised and the strychnia solution added. Cold and darkness proved valuable aids in maintaining the lives of these amphibians; the former factor for this special reason, that tetanus could be induced by lesser doses, and kept up for a longer time on the same dose in cold weather than in warm, according to my experience, thus verifying similar observations of Künde.

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*Since writing the essay, I have found that Vulpian had succeeded in this nine years ago.

Many of these frogs died on the fourth or fifth day, others, after having been in tetanus a longer or shorter time, escaped; fifteen survived to the twentieth and thirtieth day, and four lived forty-five days and longer.* There seem to be individuals of this species who have a greater amount of resisting power to the drug than others; it seems that those requiring but little strychnia and whose tetanus comes on gradually, have the best chances for recovery.

As soon as accelerated movements of the nostrils and flanks showed themselves, the strychninized water was poured off, and replaced by pure water, the tetanus following would last about twelve hours; as soon as it weakened, the strychnia solution was renewed. During the first eight days, the strength of the alkaloid had to be gradually increased, if the animal had been allowed to recover entirely from each attack; but if added while still under the influence of the earlier dose, it was kept to the same amount. After two weeks, it was found that the dose which when first given maintained tetanus twelve hours, now maintained it for two, three and even five days without renewal. I found it more difficult to maintain the tetanus during the first two weeks, than during the next two.

Frogs who escaped during the second week, and were recaptured some time after, showed no untoward symptom. No routine was adhered to strictly: the procedure was different for some individual cases.

Experiment 142.—*Rana leopardina.* November 8th; 0.001

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* Since writing the essay, I have succeeded in maintaining five frogs in tetanus for over forty days; one of these survived to the sixty-seventh day, and was intended for demonstration to the N. Y. Neurological Society, but being neglected, he was found, three days after, desiccated, having perished for want of water.

in water; four hours after flank and nostril movements spasmodic; five hours, hyoid contraction; he then occupied a drawn-together position, from which he frequently started, and the water was poured off. Tetanus developed at eight hours, with croaking; nine hours, maximal tetanus, croaking ceased, irritable; fourteen hours, recommenced croaking (augurs recovery). November 10th, A. M., animal irritable, occasional clonic shocks, pupils dilated. Strychnia renewed; four P. M., no tetanus; ten P. M., tetanus recommenced. November 10th, pupils moderately contracted; tetanus reaches an extreme degree; for six hours the animal is as rigid as a rod, and not expected to recover. November 11th, still in tonic spasm; pupils contracted. November 12th, tonic spasm occasionally gives way to clonic; croaking resumed. November 15th, A. M., still in clonic spasms, with occasional tonic spells. Strychnia renewed, P. M.; again in rigid spasm. November 14th, 15th, continued tonic spasm. November 16th, lively; relaxed, but irritable; jumps stiffly. Strychnia renewed; November 16th, full tetanus; the color of the animal is undergoing a change, the ventral yellow is becoming brighter; the cutaneous vessels are hyperæmic, especially noticeable on web. Observation imperfect up to November 27th, when the history of the 15th, 16th and 17th was repeated. November 30th, apparently dead; on opening body, heart still beating; all the internal organs, especially the omental fat-lobules and the brain well injected; the thalami and optic lobes greyer than normal; brain and medulla firm in consistency. From November 8th to November 30th, or out of 504 hours, this animal was known to have been in spasms for 298; of these, 218 hours represent one continuous period of tetanus; and it would have lived longer, doubtless, if it had not been opened under the erroneous impression that it was dead.

Experiment 246.—Middle-sized *Rana fontinalis.*

November 10th, tetanus throughout the day.
" 11th, " " " " recovering; added strychnia.
" 12th, " " " "
" 13th, " less pronounced.
" 14th, " and tremors; renewed strychnia.

November 15th, tetanus complete; added strychnia.
" 16th, " croaking; relaxation, P. M.; renewed strychnia, and did not change water.
" 17th, " throughout day complete.
" 18th, " " " "
" 19th, " " " "
" 20th, " " " "
" 21st, " " " " added strychnia.
" 22d, " " " "
" 23d, " " " "
" 26th, " anenergetic; animal stiff and extended, but continually agitated by tremors.
" 27th, " ditto.
" 28th, " ditto; tetanus relaxing; on being turned out of jar, and after passing out of the transitory tetanus, induced by the irritation, crawls clumsily, and in all its movements is decidedly ataxic.
" 29th, as yesterday; renewed strychnia.
" 30th, complete tetanus.
December 1st, " "
" 2d, " "
" 3d, " "
" 4th, recovering; added strychnia; P. M., tetanus.
" 5th, A. M., tetanus; symptoms of recovery; renewed strychnia.
December 6th, complete tetanus.
" 7th, " " renewed strychnia.
" 8th, " " tremors of a jactitating character; hyperæmia of integuments extreme.
" 9th, " "
" 10th, " "
" 11th, " "
" 12th, " "
" 13th, " "
" 14th, " "

December 15th, complete tetanus renewed; a frog of one-third the weight of the present one, immersed in the same solution, fails to show tetanus.

“ 16th, recovering; pupils in most intense contraction; on being turned out of jar, walks in a clumsy, ataxic, and halting manner, muscles rigid. Tetanus recurred before strychnia was renewed.

“ 17th, clonic spasms; strychnia renewed.

“ 18th, recovering; animal permitted to rest, in order to study its gait; every now and again the hind legs would shoot out, and when fully extended would be found relaxed, thus differing from true tetanus. In jumping the frog frequently lands on his back, although free from spasms at the time.

“ 19th, strychnia renewed; tetanus returns, of a tremulous character; pupils less contracted than yesterday, when it had no strychnia; from this time forward, it was observed that as a general thing, the increasing contraction of the pupils heralded recovery.

“ 20th, tetanus continues; still more tremulous and anenergetic.

“ 21st, Ditto.
“ 22d, “
“ 23d, “
“ 24th, “
“ 25th, “
“ 26th, “
“ 27th, “ added strychnia.
“ 28th, tetanus still more anenergetic.

“ 29th, “ still more so; the eyes have been firmly retracted for the last twelve days; the tetanus is becoming feebler, but strychnia not renewed.

“ 30th, a feeble opisthotonos this A. M.; tremors increasing in intensity; found dead and relaxed in the afternoon.

This frog was under the continual influence of strychnia for fifty-one days.

The experiment and similar ones, are important as showing that in the course of chronic poisoning certain symptoms are developed, quite distinct from those observed in acute strychnia poisoning. The anenergetic character of the tetanus, it might be argued, was due to the muscular exertion protracted beyond physiological limits, and slight tremors similar to those noticed in protracted tetanus after a single dose might be thus explained. But the tremors observed in the last stage were too excursive to be attributed to this cause, and although unable to get a full registry of the curve, satisfied myself that it was quite distinct from Kronecker's "Ermüdungs-curve." They resemble rather the results of central disease, producing active irritation. Then, also, while the animal was recovering, for a day or so on two occasions, symptoms were noticed, referable to a morbid central irritation independent of the strychnia. These are the pronounced ataxia, the "spinal epilepsy" and the pupillary contraction. In other experiments, where animals had been allowed to recover after having been in tetanus for over thirty days, the "spinal epilepsy" was observed for four and six days after the tetanic symptoms had passed away, while the ataxia and pupillary myosis continued for weeks, until the animals died, or as one did, permanently recover. I regard the "spinal epilepsy" as such, and as distinct from the tetanus not only for this reason, and on account of its character, but, also, because the spasm in the hind legs was unassociated with trunk, head, eye, and fore leg spasm, as it would have been had it been a tetanic spasm. It could be brought on occasionally by pinching the hind feet, but not by irritating any other part of the body. I should state, however, that not in all frogs are all these symptoms produced to the same extent.

Altogether these symptoms suggest central changes, and it is noteworthy that the normally white tissues of the brain should have shown in places a greyish tinge. It is to be regretted that the preservation of the brain failed, while that of the cord is not complete enough for an examination at date.*

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* Unfortunately lost, since.

Probably the deficient energy of the tetanus at the end was due to the fact that the alkaloid had but a damaged nervous axis to work on.

Experiment 253.—*Rana leopardina*, kept in tetanus nine days; death. Autopsy: arterial hyperæmia of all organs; extravasation in muscular masses of left thigh; cord and brain firm; increase of round nuclear bodies in neuroglia; nerve cells and nerve tubes entirely healthy. Brachial and sciatic nerves highly injected; no changes histologically.

Experiment 250.—*Rana leopardina*, maintained in tetanus for four successive periods of three, eight, seven and eleven days respectively, interrupted by imperfectly tetanic periods of from six to twenty-four hours. On recovering from the last dose the animal was restless for three days, then it was observed that the right eyeball protruded less than the left, the pupil of the former being in maximal contraction, the latter normal (medium dilatation). The gait became progressively more ataxic up to the fifth day; tremors and "spinal epilepsy" marked. From this time on there was a stand-still in the symptoms, the frog was indisposed for exertion, but remained crouched in a dark corner. If handled, the tremors came on. On the eighth day after ceasing administration, it died asthenic.

Besides affecting the central nervous system, chronic strychnia poisoning affects also the vaso-motor; whether this be due to the muscular over-strain, or a direct paralysis of the ganglia, I am not prepared to state. To show that like the vasomotor symptoms of acute poisoning, they are independent of the cord, I in

Experiment 287, pithed the cord of a frog, that had been in tetanus twelve days, and divided the vagi of both sides. Death seven hours after operation; injection of skin persists, no œdema.

In the case of the spinal cord of frog No. 142, I had the good fortune to discover destructive lesions of the nervous system, and thus to base the peculiar symptoms of chronic strychnia poisoning upon a pathological foundation. As might have been anticipated, these lesions were secondary results.

The specimen to be described had been hardened in bichromate of potash, after having been in a one-half per cent. solution of chromic acid; it was cut in a hand microtome and stained in carmine and hæmatoxylin, then after the usual routine, examined in clove-oil or glycerine.

The section showing the most intense changes was obtained from above the origin of the first cervical nerve; the cord here shows the commencing type of the medulla oblongata. Both posterior white columns were the seat of scattered foci of disease. There were two smaller areas on the right side, which were confluent in lower sections, and which nowhere reached the periphery; on the left side there was a single area which reached the periphery. The changes consisted in an absence of the nerve-tubes normal to the locality, their place being occupied in all three foci by a molecular and granular material, without any very pronounced structure. In addition, there was some granular and crystalline pigment present. In the right patch every transition could be observed between scarcely altered blood-discs and black angular hæmatoidin crystals. A transparent crystalline body, colorless, and with imperfect angles (possibly cholesterin). These various bodies were crowded closely together, the tissue between them was amorphous, and stained but little in carmine. Several spider cells, with marked processes, were visible in the periphery; and with these, a semi-fibrillated connective tissue, with very few nerve-tubules. This tissue contained various oblong meshes, filled with neuroglia-nuclei-like bodies in great numbers. They stained deeply in carmine.

In other segments, diffuse pigmentation, with a slight hypertrophy of the neuroglia, constituted the sole change. It could be traced up as far as the cerebellum, and down to below the roots of the great brachial nerve; beyond these points no good sections were obtainable. How far other appearances noted are to be considered pathological, I am not prepared to say, until the variations of the batrachian cord shall be better studied under normal circumstances.

Although these changes are undoubtedly the result of past hemorrhages and inflammatory stasis, and are, therefore, due to the asphyctic engorgement and later vaso-motor paresis, rather than to the local effects of the strychnia, they assume some importance when correlated with the symptoms manifested *intra-vitam.*

The white softening, which I have found in some other cases of chronic strychnia poisoning, and also in frogs dying after having been in extreme tetanus for several days, is not always the collateral softening of hemorrhages, or of oedema from engorgement. I have found it to be distinctly fascicular in its distribution; and am inclined to consider it a myelitis, the result of muscular over-exertion—such a myelitis as Leyden presumes to occur in the human subject, without exudation and hyperæmia.

The sequence of these various pathological conditions, I consider as following two lines in as many groups. First group.—Extreme tetanus, with muscular over-exertion, followed, sooner or later, by simple softening of cord; death. Second group.—Engorgement of cord, from asphyxia and strain; stasis and diapedesis of lymphoid bodies in certain foci; hemorrhage and tissue destruction in others; active hypertrophy of connective tissue in both localities, with pigmentary metamorphosis of clot in the latter; death after a considerable period, or partial recovery. All the lesions most intense in the posterior columns.

Nowhere was there any evidence that either the nerve fibres or ganglion cells were primarily diseased.

The development of spider cells, in great numbers, I have also noticed in the cord of a dog that had recovered from two strychnia intoxications, separated by an interval of eight days.* Meynert found the same appearance in the cords of hydrophobic and tetanic individuals; and it is very likely that the same appearance will be found in protracted cases of human strychnia poisoning.

The conclusion naturally follows, that there must be some factor common to all three conditions, which results in the same pathological change, and that the latter does not depend on the specific nature of the irritant. That factor is, as we have stated, the vascular engorgement.

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*It is needless for me to state that chronic strychnia tetanus is not possible in warm-blooded animals.

Summing up these results, we find, first, that frogs can be maintained in strychnia tetanus for from one to eight weeks; that at the end of the longer periods they manifest symptoms of a central affection, and that lesions are found in the cord, partly as an insular sclerosis, partly as a hemorrhagic and non-hemorrhagic myelitis, to account for those symptoms.*

Aside from their purely physiological interest, these experiments have a therapeutical one, in so far as they show that in the beginning the strychnia has to be occasionally increased, but that later it can be decreased, and given at rarer intervals, in order to keep up the physiological effects. That the pathological effects of chronic strychnia poisoning constitute a warning to those who indulge in the routine administration of the remedy, is evident; and experience has already condemned this practice.†

XI. THE RELATION OF STRYCHNIA TO ITS ANTIDOTES.

Although the question of the antidotal effects exercised by certain drugs, does not properly fall within the limits of this essay, I propose to briefly review this aspect of the question, in order to make some inferences as to the action of strychnia itself.

It is stated by some authors that hydrate of chloral, by others that the constant galvanic current, by still others that curare is an antidote to strychnia; and that cases of poisoning treated by these agents, with the assistance of quiet and darkness, can recover. The latter statement is undoubtedly true. It remains to be seen in how far this beneficial influence is truly antidotal, or whether it consists in the obliteration merely of certain symptomatic results.

Several problems enter into the discussion of this question, which complicate it considerably. In the first place, let us assume that two substances are strictly antidotal, and will in given relative doses, neutralize each other's effects entirely. In order to do this, it is evident that their administration must be so timed (at least with regard to neurotics), that the height of the effect of the one drug will be synchronous with the height of the effect in the other. These conditions it is almost impossible to comply with; and it may happen that instead of neutralizing each other, the toxic effects of two alkaloids may virtually be added together.

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* A proliferation of the epithelium has been noted around the central canal in one specimen.

† As an example, I may quote the case of a lady seen by myself, in consultation with Dr. Skinner, of this city, in which I made the diagnosis of a tumor, pressing upon the cord at the vertebra prominens. Here as little as $\frac{1}{100}$ of a grain of strychnia produced intense aggravation of the symptoms before I saw the case.

I have found that chloralized rabbits show the toxic effects of large doses of strychnia as rapidly as non-chloralized animals; that moderately large doses produce tetanus in spite of chloral, although the effects of small doses are annulled. My experience with the galvanic current is the same.

It is, therefore, my opinion that these and other antidotes act favorably by suppressing certain of the results of strychnia at the periphery, but that they do not neutralize the essentially lethal action of the alkaloid, which it exercises on the nerve cell protoplasm. *Per se*, strychnia kills the nerve cell, as it is able to kill other complex protoplasmic bodies (leucocytes).

We may state, that the galvanic current alters the electrotonus of the cord, so as to prevent the transmission of the tetanizing influence to the periphery; that curare paralyzes the intra-muscular nerve-ends, and renders it impossible for the muscle to respond; and that chloral blunts the sensibility and counteracts the transmission of centripetal irritations; but we cannot assert that any of these agents neutralizes the central influence of strychnia. The drug which, in my opinion, approaches most closely to the ideal of an antidote, is conium.

The three remedies first mentioned, are no more true antidotes than are darkness, quiet, and a moderate temperature, or any other suppression of peripheral irritation. I should add, however, that because irritation provokes strychnia spasms, it does not follow that without irritation there will necessarily always be fewer spasms. When large doses are given to two frogs of the same size, the one frequently irritated will cease to respond, after a time, altogether; such spasms as occur then, come on spontaneously. In the other, the spasms will be fewer at first, and progressively become more intense, frequent and permanent. Both will die at about the same time, and have had the same sum total of spasms.

The therapeutical agents mentioned have no influence in abolishing the sequels of chronic strychnia poisoning.

In proposing remedies for strychnia poisoning, the tetanus has chiefly been regarded, and sight lost of the fact, that strychnia in large doses is an essential poison to the nervous system, whether convulsions occur or not.

SUMMARY.

1. Strychnia is a poison to all forms of animal life.

2. Strychnia exercises its influence upon animals by affecting the central nervous apparatus and the vaso-motor system, but it can act fatally on animal forms possessing neither of these systems, and must therefore be considered as acting lethally on all complex tissues.

3. Strychnia acts on the grey matter, on all grey matter alike, and affects both its sensory and motor elements.

4. The white nervous matter and the peripheral nerves play but the passive rôle of conductors.

5. Strychnia does not affect the nerves or muscles locally.

6. It affects the peripheral end organs of special sense locally.

7. Strychnia produces tonic spasm in all vertebrates.

8. In higher vertebrates, clonic spasms are added.

9. This difference between higher and lower vertebrates in this respect is probably due to the difference of the nervous system.

10. The clonic spasms are more intense, *cæteris paribus*, in smaller than in larger animals.

11. In higher animals the spasms are due to the action of the alkaloid on the axial parts of the nervous system, the pons, medulla and cord.

12. The maximum of the tetanizing influence is intra-cranial, and thence it gradually diminishes toward the posterior extremity of the cord.

13. Strychnia kills the lower vertebrates directly by nervous exhaustion, the higher, as a rule, by asphyxia and venous congestion of the nervous system in addition. In very large doses it is directly lethal to the nervous tissues.

14. Its vaso-motor effect is to increase the blood pressure, and the rapidity of the blood current, by contracting the arterial vessels. This effect is independent of the central nervous system.

15. Its effect on the heart is to augment the systole and prolong the diastole; the heart may cease acting by tetanic rigidity in any stage. These effects are produced first by the action of the alkaloid, on the local ganglia of the heart; secondly, through the pneumogastric nerve.

16. In large doses, cortical (epileptiform) spasms are presumably produced.

·17. Strychnia is present in every organ of the body, after administration; it is found in all parts of the brain, cord and nerves.

18. It is in greater quantities in the grey than in the white nerve tissues.

19. It produces no visible changes either in the nerve fibres or nerve cells, whether given for a short or a long period.

20. Lesions are found after death from strychnia, which are not due to its direct local effect, but to secondary results.

21. Chronic permanent lesions are produced in the course of chronic strychnia tetanus, which are also referable to secondary influences.

22. There is no true antidote to strychnia, although various agents which diminish reflex excitability, and paralyze the muscles, or alter the tonus of nerve fibres, may enable an animal to survive the effects of otherwise fatal doses. But these means fail with larger doses.

23. Strychnia increases the reflex excitability, but not all strychnia spasms are due to this factor: some depend upon direct irritation of the motor cells.

24. As with other irritant toxic agents, the lethal effects are preceded by a stage of functional stimulation.

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Art. II.—OTHER SYMPTOMS OF NEURASTHENIA (NERVOUS EXHAUSTION).

By Geo. M. Beard, M. D., of New York.

In a paper read before the New York Academy of Medicine, and published in the *Virginia Medical Monthly* for June, 1878, I described certain symptoms of Neurasthenia (nervous exhaustion) as follows:

Tenderness of the scalp (cerebral irritation, cerebrasthenia); tenderness of the spine (spinal irritation, myelasthenia); tenderness of the teeth and gums; tenderness of the whole body (general hyperæsthesia); general or local itching; abnormalities of the secretions; vague pains and flying neuralgias; flushing and fidgetiness; tremulous and variable pulse with palpitation; sudden giving way of general or special functions; special idiosyncrasies in regard to food, medicine, and external irritants; sensitiveness to changes in the weather; a feeling of profound exhaustion unaccompanied by pain; ticklishness; desire for stimulants and narcotics; insomnia; nervous dyspepsia; partial failure of memory; deficient mental control; seminal emissions; spermatorrhea; partial or complete impotence; changes in the expression of the eyes and countenance; mental depression, with general timidity; morbid fear of special kinds, as agoraphobia (fear of places); astraphobia (fear of lightning); sick headache and various forms of headache; disturbances of the nerves and organs of special sense; localized peripheral numbness and hyperæsthesia; general and local chills and flashes of heat; local spasms of muscles.*

The above list is not only not exhaustive, but a number of

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*Some of these symptoms, but not all of them, were described in my original paper on "Neurasthenia," published in the *Boston Medical and Surgical Journal*, April 29, 1869. I may refer also to a paper on "The Nature and Diagnosis of Neurasthenia," in the *New York Medical Journal*, March, 1879; and to Erb's chapter on the same subject, in volume XIII of Ziemssen's Cyclopedia.

the phenomena embraced under the various heads have been described thus far but incompletely.

I purpose, in the present essay, to supplement the clinical picture of this very frequent and little studied nervous disease by analyzing somewhat more in detail some of the symptoms already noticed, and by describing others that, so far as I know, have not been previously described, at least, not in their relation to neurasthenia.

Some of the above symptoms, when they are mentioned at all in works on diseases of the nervous system, have been and now are referred to under such headings as cerebral anæmia, or hyperæmia, or general anæmia, or hysteria, or hypochondriasis, or oxaluria; and some of them are mentioned in connection with structural lesions, as ataxy and muscular atrophy, and by many are regarded as essential parts of the clinical image of these grave disorders.

DEFICIENT THIRST AND CAPACITY FOR ASSIMILATING FLUIDS.

Quite recently a physician who consulted me in regard to himself, called my attention to the highly interesting fact that he rarely drank water either at meals or between meals; and he stated that the average quantity of liquid that he consumed was far below the normal standard. Investigation of other cases of neurasthenia has convinced me that this deficiency of thirst is one of the symptoms of that state, and it would appear that it is not an unusual symptom, but exists in not a few cases; there are many who for years have a poor appetite for fluids as they have a poor appetite for solid food; they live on a small quantity of liquid, and, perhaps, without suspecting it until their attention is directed to the fact. There are those who find that if they take much liquid the stomach suffers even when little or no solid food is mingled with it. One advantage, with some disadvantages, of the free use of beer with our German friends is in the quantity of fluid that they thereby imbibe—the water of the drink more than the alcohol. Drinking milk has a similar advantage.

When we remember that the body is composed mostly of water, we can easily see that there is a danger of starving for want of simple liquid, just as, under the influences of our civilization, we are starving for want of fatty food.

There is no question that the Europeans, who are far less nervous than the Americans, use far more liquid nourishment; and it is also a fact, more and more impressed on my mind, that many neurasthenic patients are very temperate, if not total abstainers, and some abstain even from tea and coffee.

ABNORMAL DRYNESS OF THE SKIN, JOINTS AND MUCOUS MEMBRANES.

In some cases of neurasthenia the skin of the whole body is unnaturally dry; this is especially and most readily noticed in the hands, but all parts of the surface may present this peculiarity. A scaliness or scurfiness may accompany this dryness, as though there were a deficiency of fluids and of sebaceous secretion. There would also appear to be a relation between this condition and a disinclination to drink, or use fluids freely.

A young lady of twenty-one, in addition to many other neurasthenic symptoms, had dryness of the scalp, eyes and ears, especially in the morning; her eyes and ears would be dry and hot, and in the ears a thin skin would form, and fine scales would be thrown off. These symptoms were not constant; they would leave entirely for a number of days, and then, without any apparent cause, return—thus following the law of all other symptoms of neurasthenia.

Dryness of the joints is also observed in the nervously exhausted. How the joints may suffer in grave spinal diseases, such as ataxy, is well known; but in these functional maladies of which neurasthenia is a type, the joints may suffer, though in a less severe manner. Deficiency of the secretion, with dryness and cracking sound on movement, I have noticed in a number of cases; in one striking case of musician's cramp, cracking of the joints is noticed in the affected fingers.

SWEATING HANDS, WITH REDNESS.

Sweating of the hands—of the palmar surface, or of the entire hand—palmar hyperidrosis— is a symptom of neurasthenia at once so interesting and so frequent, that I wonder that the literature of the subject is so meager.

Very frequently indeed this symptom is one of the results and accompaniments of sexual debility, especially when caused by masturbation; but it can hardly be regarded as diagnostic of sexual exhaustion, nor would I, on that fact alone, decide that the genital system was primarily at fault. This phenomenon—abnormal perspiration of the hands—is certainly more common in males than in females, although it occurs, as, indeed, all forms of hyperidrosis occur, in both sexes. The milder phases are common enough, but there are severe manifestations that this symptom may assume, which seem well nigh beyond belief. Thus a young man now under my care is so distressed thereby that he threatens suicide unless he is permanently cured. In his case there are various evidences of a bad inheritance, a poor constitution, although this palmar sweating is just now the only very annoying expression of the depraved diathesis. A young lady in the northern part of the State is compelled to take a number of handkerchiefs with her when she goes to school, and on her return they are all saturated from the excessive perspiration of her hands. My friend, Dr. Josiah Roberts of this city, tells me that in a similar case, lately brought to his attention, there was clear proof of uterine disease.

The intimate relation of this symptom to the nervous system is shown in many striking facts. Thus one young man who consulted me would be attacked periodically—at ten and four o'clock—and whenever he was at sea the symptom would utterly leave him. In one of my cases the slightest emotion would instantly saturate the hands as thoroughly as dipping them in a pail of water. The effort to shake hands is sufficient to produce this effect. Redness of the whole hand—erythema—sometimes attends this palmar sweating, and in one of my cases the ears are as red as the hands.

CONVULSIVE MOVEMENTS, ESPECIALLY ON GOING TO SLEEP.

Nervous sufferers, just as they are dropping off to sleep, are sometimes suddenly and painfully awakened by a violent, spasmodic movement of an arm, or leg, or of the whole body. This appears without any warning, and is most likely to occur when preceded by unusual excitement or fatigue. In some cases there will be a recurrence of these spasms, so that much difficulty is experienced in getting to sleep. I have known instances where the whole body seemed to be thrown off the bed, or rather, the sensation was as though the body were projected upward. This symptom is not so alarming as some of those who experience it believe. It indicates an exhausted, a worn condition of the nervous system; but it is not as ominous for evil as many other phenomena that belong to the nervously exhausted state. A friend of mine—a public speaker, constantly before audiences, and always at work—with a frame of unusual size and an extraordinary capacity for enduring mental excitement and toil—tells me that with all his vigor he has been annoyed by these jerkings on falling to sleep, although he has no other evidence of neurasthenia.

It is probable that these convulsive symptoms on dropping to sleep are the effect and sign of congestion in the exhausted nerve centres, and occur while passing out of the waking into the sleeping condition, because the inhibitory or controlling power of the waking state is removed.

ATONIC VOICE.

When neurasthenia lays its hands on a man it is liable to leave its impress on every organ and function of the body; from the crown to the toe there is not a fibre that is safe from attack. If some parts escape in one individual, they suffer in others. If at one stage of the malady certain regions are unaffected, it may be only that they may be attacked with all the greater violence at another stage. Thus the hair, the scalp, the eyes, the ears, the nasal and respiratory passages; the brain, in whole or in part, the cranial nerves, the heart, the spinal cord in any portion, the sensory and motor nerves, the stomach and bowels, the reproductive system, the skin, the nails, the secretions, the excretions, the absorbents—all are objects of assault.

It is not strange, therefore, that there should be a neurasthenic voice, just as there is a neurasthenic eye, a neurasthenic stomach and genitals. The chief peculiarity of the neurasthenic voice, is softness, faintness, want of courage and clearness of tone. These terms, though vague, express perhaps, as well as it is possible to do in words, how this voice deviates from the normal voice, but at best verbal descriptions are faulty, and far inferior to even a single living illustration. To a physician accustomed to see these cases and to observe the voice, there is but little difficulty in at least suspecting the diagnosis by this symptom alone. This neurasthenic voice somewhat resembles the peculiar voice of the deaf; and yet it is not precisely like that, and can usually be distinguished from it. A neurasthenic sufferer may have the muscle of an athlete, and be so strong that a hard day's toil is but play, and yet speak in a voice which in quality and volume of sound suggests the beginning of convalescence from a severe fever.

"The voice," says Emerson, "is a delicate index of the soul," and with scientific truth the same philosopher asserts, that the orator can often tell by the quality of his own speech, at the beginning of an oration, or sermon, whether he is or not in the mood of speaking, whether he is to be eloquent or utterly fail.

A dissolute life, especially in women, always registers itself in the voice, impressing a coarseness that in its quality is almost diagnostic. The queens of song are never grossly impure.

OXALATES AND URATES IN THE URINE.

The relation of oxalate of lime to various nervous symptoms was long ago pointed out by Golding Bird, and the importance of examining the urine for the deposits of the oxalates was strenuously insisted on by him; but the true relation of such deposits to the nervous system seems not to have been fully understood either by him or by those who have since written on the subject.

As a matter of routine I have, for years, been accustomed to have the urine of my neurasthenic patients examined, and in the majority of cases it is found that the oxalates, and in some cases, the urates, are in great excess. Amorphous urate deposits are noticed, also uric acid crystals.

The term "oxaluria," so often applied to this condition, is quite analogous to the term "spinal irritation," as applied to tenderness of the cord with accompanying symptoms, so often observed in neurasthenia. To the employment of such terms there can be no objection, provided those who use them understand that in scientific strictness they do not mean disease of a distinct character, but are really results and expressions of neurasthenia, mal-nutrition of the nervous system. The urine of the neurasthenic is often, if not usually, abnormally acid; and spermatozoa are frequently found even when there is no special reason on the part of the patient, or of the physician, to suspect any marked degree of seminal weakness. In a philosophic sense these oxalates and urates, and the acidity and spermatozoa, are like spinal irritation, cerebral irritation, neurasthenia, asthenopia, and dyspepsia, results, effects, in a word, symptoms, and if the cause at all of other symptoms, are but secondarily so.

GAPING AND YAWNING.

As evidences of temporary fatigue, gaping and yawning are familiar enough even though their physiology may be obscure. In organic disease of the brain also, frequent and prolonged gaping has been noticed.

In one case of glosso-labial paralysis that I saw a number of years ago, this symptom of gaping was so frequent, and the act so prolonged, as to be ludicrous.

In neurasthenia, gaping, yawning, and stretching may appear like the other symptoms mentioned, and like them also the attacks come and go; they are quite apt to follow over-exertion or excitement, even when there has been no loss of sleep. A neurasthenic patient, now under my care, tells me that after long reading a newspaper in the morning after breakfast, he is troubled with gaping, though no other evidence of weariness annoys him; in his case the eyes are asthenopic, and prolonged use sometimes brings on various symptoms.

DILATED PUPILS.

Dilatation of the pupils is so often seen in neurasthenia, especially in the sexual forms, that it may be considered as an important fact to be noted in the study of a case. I do not look upon this symptom as in any sense diagnostic, although it is so often associated with a history of genital disturbance, for there are cases of sexual exhaustion where it does not exist, and it sometimes exists where there are no proofs of sexual trouble.

Abnormal activity of the pupil—sudden and frequent alternations between contraction and dilatation—is a sign of neurasthenia, or, at least, of nervous irritability, of perhaps more importance than mere dilatation, just as in organic diseases of the cord, sluggishness of the pupils, slowness to contraction or dilate, has been recently suggested as a better diagnostic sign than mere contraction of one or both pupils.

SHOOTING PAINS SIMULATING THOSE OF ATAXY.

If there be any difference between the familiar shooting, lightning-like pains in the extremities that have so long been considered as peculiar to ataxy, and the shooting pains of neurasthenia, I have not been able as yet to find it out. Generally these neurasthenic pains are milder than those of ataxy, but this average fact does not interfere with the fact of observation, that this difference in degree is not of itself sufficient to make it possible to establish the differential diagnosis; for the shooting pains of ataxy are by no means always severe, and in many cases of the disease do not exist at all. The mistake of writers in so strenuously insisting on the diagnostic importance of these shooting pains has been, and is, the source of terrible annoyance to physicians, especially who happen to be themselves sufferers from these neurasthenic symptoms.

The same remark applies to fibrillary contractions, which have been looked upon as indicating muscular atrophy, but which, as I have elsewhere stated, may consist of one of the many symptoms of neurasthenia.

PECULIARITIES OF PAIN IN THE BACK.

In neurasthenia all parts of the back may be the seat of pain, although certain districts are more affected than others. There may be tenderness when there is no pain, and conversely pain, even severe pain, when there is no tenderness. There may be much distress in the loins and over the hips, when careful examination shows no tenderness anywhere.

This pain in the hips and loins is something quite different from ordinary neuralgia or sciatica; it rather resembles muscular rheumatism or a common cold, and is indeed often confounded with one or both of these diseases even by able diagnosticians. The liability to confound irritation of the upper part of the spine at the nape of the neck with rheumatism is very great; the symptoms, indeed, are quite the same —pain, stiffness, aching, inability to move the head without discomfort. Sometimes this condition perfectly simulates wry neck, and is mistaken for it. One of the very ablest neurologists in Germany, on being consulted by a case of irritation in the upper part of the spine, made diagnosis of rheumatism, and treated the patient accordingly. This back pain, and the tenderness that may or may not accompany it, fluctuates like all these neurasthenic symptoms; to-day they are present in full force, to-morrow they are all gone, but, on any provocation, are liable to return. They fly about in every direction; now just below the shoulder-blade; now in the centre of the spine; and at another time between the shoulder blades, or in the middle lumbar region; sometimes with heat and burning, at others with biting, penetrating sensations, or a feeling as though ants were crawling just under the skin.

HEAVINESS OF THE LOINS AND LIMBS.

One of the most frequent complaints among the neurasthenic (myelasthenic form) is heaviness and vague aching of the loins and limbs, and sometimes of the whole body. This is a symptom hard to define in exact words, but it is very common, and it is a cause of great distress. This symptom is quite apt to follow over physical exertion, as in walking or standing, but may come on without any apparent or special exciting cause. This feeling so closely resembles rheumatism that it is often confounded with that affection by those who are unfamiliar with neurasthenia, and even one well acquainted with nervous exhaustion in all its forms, might, on first being called to a patient, mistake this heaviness and aching for a common cold, or for a rheumatic attack.* I have lately been consulted by a gentleman suffering from myelasthenia, where this aching of the lower part of the back and loins are almost the only subjective symptoms. In sexual exhaustion pains in the loins and limbs, not amounting to neuralgia but sufficient to be a severe annoyance, are frequent enough, but they are not restricted to the sexual variety of neurasthenia.

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*How the symptoms of ataxy have been, and are, mistaken for rheumatism is well known to the physician.

VARIETIES OF MORBID FEAR (PHOBIA).

There would seem to be almost no limit to the phases that morbid fear may assume in the nervously exhausted. These varieties of diseased apprehension have been observed for years, not only in the positively insane, but in the so-called hypochondriacal and melancholic; but they have not been regarded as worthy of careful scrutiny and analysis; the patients who complain of them have been dismissed, as merely imaginary, and the many accompanying symptoms which, when thoroughly studied, would have shed much light on the condition that gives rise to these morbid fears, have likewise been passed by.

Recently, however, science has endeavored to bring these vague phenomena into the realm of systematic observation, analyzing their minute manifestations, showing their relations and dependencies, and giving them, in certain cases where their frequency and positiveness would warrant, distinct names; thus astraphobia—fear of lightning—was described by me some years since; agoraphobia—fear of places—has been described by Westphal, of Berlin; and, still more recently, I have applied the term anthrophobia—fear of society—to that morbid apprehension of going into company, or of encountering human beings in any relation, which is so often seen in the nervously exhausted, especially in those sexually exhausted. One of the many phases of anthrophobia is inability to look in the face of one with whom we are conversing; this is seen frequently in sexual exhaustion, as all students of that form of nervous disease know; but it is no more restricted to that type alone than are any of the other symptoms that have been cited. Indeed, any form of exhaustion may give rise to any form of morbid fear, although, so far as I can judge from my own observation, sexual trouble—masturbation and excess—is at least one of the prominent factors in the majority of cases.

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The general philosophy of this morbid fear in the neurasthenic (cerebrasthenia) would appear to be that the debility of the brain—the nerve impoverishment—renders it impossible to meet responsibility, just as paraplegia makes it difficult or impossible to walk; morbid fear is indeed but a psychical paralysis, but of a functional rather than of an organic nature.

The world over, aversion of the eyes with a turning away of the face, is an expression of the emotion of humility and bashfulness, that is, of a feeling of weakness as compared with the person in whose presence we stand—an instinctive and involuntary recognition of the fact that, for the moment, our force is inferior to his. In neurasthenia this same principle appears as a pathological symptom—an expression of debility, of inadequacy, of incompetence. This aversion of the eyes is so constant a symptom in these neurasthenic patients, that I often make the diagnosis as soon as they enter the office, before a word has been spoken by either party, and even before the patient has had time to be seated. I have now under my care a young man with sexual exhaustion, who is so badly anthrophobic, that even when I take his head in my hands and hold it up, it is impossible to keep his eyes fixed on mine for more than an instant. A very intelligent and able friend now under my professional care, displays this same characteristic, and I have often talked with him in regard to it.

Some of the phases of this morbid fear are very interesting and surprising, even to those who are most familiar with the caprices of the diseased nervous system. I have elsewhere published a brief account of the physician who consulted me in regard to himself for long-standing cerebrasthenia, one of the symptoms of which was inability to go away from his home or office, or place where he was stopping, to any considerable distance in a direct line. He had the muscular strength to walk twenty miles, but when summoned to a patient was often obliged to decline to attempt to go even half a mile, which was a great astonishment to his patients, who were aware that even when unable to visit them he could work all day in his garden. Like many of these cases he had a morbid fear of visiting the place where he was first attacked by any of his ill feelings; thus he had been at one time prostrated in New York city, and felt incompetent to come here to consult me; accordingly I met him by appointment in a distant city. In walking out with him one morning I observed that he continually turned off to the side streets, so as to keep at a little distance from the hotel where we were stopping for the day, and, on my questioning him, he said that he could not go more than half a mile in a straight line, and that therefore he turned into the side streets so as to keep the hotel near at hand; the result was that we walked arm in arm, circumnavigating the hotel at a moderate distance—although not always keeping it in sight. The patient was not at all wearied, although the walk was a long one—in a direct line perhaps a mile or two.

I have now under care a patient whose morbid fear takes just the opposite phase: he cannot go to a certain locality, but can go very near to it, and beyond that point his own will is often powerless to urge him forward. He was first attacked while in a lithographic establishment working at his trade, and from that hour he has found it hard or actually impossible to enter any building devoted to that business. One day he resolved that he would conquer what seemed to him and his friends a foolish whim, and started out for the shop, but on arriving within sight—about the distance of a block—he was compelled to stop; a cordon of policemen could not have been a more effective blockade; resolved not to be beaten, he retired a short distance, and approached the building from another direction, but was again brought up against the imaginary barrier, and so in succession all the points of the compass were tried with absolute failure.

He had a chance to work in Syracuse, and went to the depot to take the train for that city, but on entering the station and going up to the office, he burst into tears, and could not buy his ticket; he tried and tried, and finally gave up and returned home. He could have walked to Syracuse, but he could not reach out his hand and purchase the ticket for his fare. At another time he succeeded in reaching Cincinnati in quest of employment, and was directed to a lithographic establishment where he expected to be employed; but in spite of all his repeated trials he could only come within sight of the building, and he was forced to return to New York.

I have just been consulted by a physician who, as one of the effects and signs of cerebrasthenia, cannot at times undertake any slight responsibility; thus he has sometimes allowed a large number of horse-cars to pass him before he could bring up the resolution to jump on board one of them; and yet his muscular strength at the time was excellent.

Inability to travel alone is one of the myriad forms of morbid fear, and is indeed quite the opposite of anthrophobia, just described. During the past year two cases of this kind have been under my observation—one a clergyman, the other a merchant, and both competent to attend to their respective duties: the first as teacher in a responsible position, the second as partner in a mercantile house; although both suffer, and, for years, have suffered from very many other symptoms of neurasthenia.

HOPELESSNESS.

When a patient is dying in the last stages of consumption or cancer, he is often, if not usually, hopeful; and sometimes he does not abandon the expectation of recovery even when on the edge of the grave. After friends have given up utterly, and the physician only comes to relieve, the patient himself is full of hope.

In functional nervous disorders, that are relievable if not curable, the reverse phenomenon is observed. The patient, even in the earlier and milder stages, is without hope, while the friends laugh at his fears and ridicule him for talking or thinking of his symptoms. A good example is found in an attack of sick headache, but nearly all the neuroses exhibit this phenomenon, in greater or less degree; as for instance, writer's cramp, hay fever, musician's cramp, telegrapher's cramp, and the malady here under consideration, neurasthenia, in its various phases.

In organic, structural and incurable disease, such as cerebral paralysis, paraplegia, etc., the sufferer is far less likely to despair of relief.

The philosophy of this symptom of hopelessness appears to be similar to that of morbid fear, as above described. That is, an instinctive consciousness of inadequacy for the task before us. We are hopeless because our nerve force is so reduced that the mere holding on to life seems to be a burden too heavy for us. A certain amount of nerve strength is necessary to supply the courage requisite for simple existence. Abstaining from dying demands a degree of force just as the mere keeping in an erect position—standing up without taking a single step—is only possible to those who have a certain quantity of strength. Abstaining from dying, like abstaining from falling, is in one respect a negation only, but neither is possible without an expenditure of force.

The despair of sea-sickness well illustrates this phenomenon. In the short space of an hour, or less, one can be reduced from a state of perfect bliss to perfect misery, simply from the perturbations caused by the motion of the vessel.

One time, when returning from England, our steamer collided with a sailing vessel in such a way and under such circumstances as to give just reason for the belief that we might be in serious peril. In the height of the excitement and alarm a sea-sick passenger came out from his room, where he had been shut up ever since our departure, and inquired what the trouble was all about. He was informed that our steamer was leaking and that we were fast sinking. "If that's all, I'll turn in again," he replied, and went back to his berth, whence he did not emerge until we all landed in New York.

In some cases of neurasthenia this hopelessness is intermittent, periodic, like attacks of inebriety or neuralgia, and these attacks are quite independent of all external conditions, although they may be excited and modified more or less by the environment.

PECULIARITIES OF INSOMNIA.

The different phases of insomnia in neurasthenic patients are exceedingly interesting.

One man finds no difficulty in getting to sleep on retiring, but soon wakes, and must remain awake for the rest of the night. Another man rolls and tumbles for hours before he falls into oblivious slumber, but when once asleep does not usually wake until morning. I was recently consulted for a case of insomnia of many years' duration, where there had never been any difficulty in sleeping after getting to sleep.

Other sufferers report that they sleep in fragments—oases of repose in a desert of dreary wakefulness—but bad dreams constantly harass them so that in the morning they are less rested than they should be. Why a bad dream should be a bad symptom is not quite clear. Why a man disturbed by indigestion or exhausted nervously by excitement late in the evening, should dream of snakes and monsters instead of green fields and gardens, of death and murder instead of delightful society and experiences, has perhaps only this general explanation, that the normal action of the cerebral cells is designed to be, in the main, pleasurable, and that mental, like physical pain, is a symptom of something abnormal. It is also a question how far dreams are pathological. It would seem that in perfect health—if there be such a state—one might dream even unpleasantly; and yet there is no doubt that savages, and farmers, and, in general, those who live out-doors, depending on their muscles for their subsistence, dream far less than the in-door brain workers. My guide in the woods of Maine and northern New Hampshire, tells me that he very rarely dreams, and one cool, phlegmatic man, whom I met in that region last summer, assured me again and again that he never, in all his life, had a dream that he could recall; and with that class, as a rule, dreams of any kind, good or bad, are exceptional.

Some neurasthenic patients can only sleep by night—never by day, however wearied. Others can sleep by day; often fall to sleep when they especially desire to keep awake, but at night toss in painful activity.

Physical exercise also acts very capriciously with different persons. Thus one of my patients tells me that if he takes a long walk in the evening, he is more restless than usual that night; and yet he is a very strong man, capable of much physical and mental toil.

APPEARANCE OF YOUTH.

Persons afflicted with neurasthenia, very often, and I think in the majority of cases, where the condition is constitutional and long-standing, look younger than their years; they bear the weight of time more easily than the phlegmatic and the strong; and when between, say thirty-five and forty-five, will pass for five or ten years below their actual age. I have reached this generalization not hastily, but after much observation and reflection. Constantly I find myself astonished when a new patient, whom I have never before seen, tells me his age. I observe that those who have had a long battle with their morbid feelings, who have been perhaps disabled, crippled, exiled by nervous incapacity, look ten years younger than their vigorous friends. The neurasthenic are, as a rule, less wrinkled and worn ; they have less fat and muscle that furnish the materials for flabbiness and coarseness of feature. Their skins are thinner and softer, and show the blood more readily. They are also less likely to be attacked with those degenerative changes in the blood-vessels and the skin, that are the signs and results of age. In a word, they look young for the same reason that they live long.

There is a still wider generalization that can be verified—namely, that the nervousness that attends civilization is everywhere accompanied by this appearance of youth. The higher classes look younger than their years, the lower classes look older than their years. Some time since, when I was connected with the Nervous Department of Demilt Dispensary, New York, I noticed that the majority of the patients looked from five to ten years older than they were. This was true of both sexes, and in nearly all forms of nervous disease. Those between twenty and thirty appeared to be thirty-five or more, and only repeated questioning in some cases would convince me that there was not either ignorance or deception. But scarcely any of these patients were neurasthenic, for in that class neurasthenic and allied affections are very rare.

ART. III.—SAPONIN, IN ITS RELATIONS TO PHYSIOLOGY.

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[Dr. Lautenbach's paper was written more than a year ago. Some more recent researches of his are added in the foot notes in brackets.—EDS.]

IN the present essay it is designed to treat in a brief manner of the physiological properties of a principle existing in a number of plants indigenous to America, which has, in the author's hands, yielded some interesting and important results. Important, in that it gives the physiologist a new agent to study certain of the functions of living tissues, which have hitherto been beyond his reach. Interesting, in that its action on certain tissues resembles that of no other known substance.

The glucoside saponin exists in almost all the caryophyllaciæ,* in all the agaves, as also in the *Polygala senega* and in the *Yucca baccata.*† It was first obtained by Schrade from the common soapwort (*Saponaria officinalis*). Shortly after Schrade, Bley‡ obtained it from the same plant. Later, Bussy§ found it in the oriental soapwort (*Gypsophila struthium*). It exists most abundantly, as was shown by Fleury and Boutier-Charland,‖ in the Brazilian soapbark.¶ Malapert determined its existence in the ovaries and fruit of the horse-chestnut.

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*Among others in the *Gypsophila struthium*, *Lychnis diœceæ*, *L. chalcedonica*, *Saponaria officinalis*, *Agrostemma githago*, and *Siline inflata*.

†A recently discovered plant from southern Arizona, which is used by the inhabitants as a substitute for soap.

‡*Ann. Chem. et Pharm.*, IV., p. 283.

§*Ann. Chem. et Pharm.*, VII., p. 168.

‖*J. Pharm.*, IV., p. 249.

¶*Quillaya saponaria.* The bark of this evergreen tree has been used in the arts to remove grease-spots from cloth.

[The glucoside chamœlerin, discovered by Dr. Green (*Amer. Jour. of Pharmacy*, 1878), I have found to act physiologically in all respects like saponin, which it closely resembles.]

As usually obtained, saponin consists of a greyish-yellow powder of a sweetish taste, inodorous in the powdered condition, very soluble in water, but sparingly soluble in dilute alcohol, and entirely insoluble in absolute alcohol and ether. The watery solution froths like soap, and it is from this property that the substance gets its name. According to Rochleder and Schwarz its chemical composition is $C_{12} H_{20} O_7$. It is readily precipitated by soluble lead* and barium salts, and it to a slight extent reduces cupric oxide in the presence of an alkali.

Quevenne† determined this glucoside to be chemically identical with the polygalic acid from *Polygala senega*, and also that the physiological properties of the two substances were the same. The author's results on this point coincide with those obtained by this distinguished chemist.

GENERAL ACTION.

No cases of saponin-poisoning have as yet been reported, and we have to rely for our knowledge of the action of this substance on the human animal, on the reports of cases of poisoning from plants containing it. These are by no means rare in the European scientific and legal records of a third of a century ago. Numerous instances were then reported where agrostemma seeds were ground with the wheat in the preparation of flour, the ingestion of which produced poisonous symptoms. Two cases of poisoning in this manner were observed by Bonneau and Malapert,‡ and five cases came under the observation of Bellard.§

The symptoms observed were: general malaise, headache, vertigo, great difficulty in maintaining the upright posture, vomiting, increased secretion in the air passages, a hot skin, and a rapid, small pulse. In the more serious cases coma occurred, from which the poisoned person could only be aroused by very powerful irritants. After death ulceration of the stomach and increased redness of this organ was observed.

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*On this account care must be taken in making a solution of saponin to use pure water, as water which has been standing in lead pipes frequently forms a precipitate.

†*Journ. de Pharm. et des Sciences Accessoires*, Sept., 1836, p. 449.

‡*Ann. d'hygiene Publ.*, XLVII., Avril, 1852, p. 350.

§*Casper's Vierteljahrschrift*, Bd. II., p. 100.

If in other vertebrates saponin is introduced into the alimentary canal, the same symptoms are produced. In addition, however, there occurs an increased frequency of the respiratory movements, bloody fæcal discharges, and convulsions.* After death the stomach and intestines are almost always found reddened and softened, and at various portions of the alimentary canal extravasations of blood are found. The lungs are frequently in a hyperæmic state.

LOCAL ACTION.

On the skin.—One drop of a solution of saponin (1 gramme to 10 c. c.) applied to the unabraded skin, produces an unpleasant feeling, not amounting, however, to absolute pain. At the same time the skin, at the point of application, becomes more sensitive. This hyperæsthesia is sometimes accompanied by redness.

On the abraded skin the application is very painful, and is often followed by pustules. These latter are, however, limited to the portion of the skin to which the solution was applied. Redness is an invariable result of such an application.

On the mucous membranes.—On these the drug acts as a decided irritant. Inhaled into the nostrils it produces sneezing, and even painfully makes its presence known to the experimenter. At the same time a decided increase in the secretion of nasal mucus occurs.†

Applied to the *mucous membrane of the mouth* its original sweetish taste is soon followed by its irritant effects. These are manifested in an increased flow of the saliva, irritation of the fauces, accompanied by a scratching sensation which causes the person or animal to hawk for a long time after the application. The taste at the same time becomes burning or biting.

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*The author has observed the ingestion of saponin to produce convulsions most frequently in birds, but they also occur in rabbits, cats, mice and dogs. In the dog the symptoms of poisoning above given occur only in exceptional instances, owing to this animal vomiting the drug.

†It is doubtless that powdered Quillaya bark owes its efficiency in cases of coryza to this property.

When by means of a stomach tube a solution of the drug is directly introduced into the *stomach*, these irritant properties become even more manifest. The animal retches continually; the respirations become more frequent, as does also the beat of the heart. In a short time vomiting results in dogs and cats; but even after this the symptoms of irritation continue for some time. If five grammes were thus introduced the animals frequently died from the gastro-intestinal irritation. After death the stomach and intestines were not only found reddened and softened, but were also the seat of numerous blood extravasations.

Inhaled into the *lungs* saponin produces marked irritant effects—coughing, hawking, etc.—accompanied by an increased mucus discharge from the air passages.*

On the skeleton muscles.†—Pelikan‡ found that, when he injected "1-2 gtt. of a syrupy solution of saponin" into the cellular tissue of a frog's leg, near the insertion of the gastrocnemius tendon, complete paralysis of the adjacent muscles was produced. Direct electrical irritation of the poisoned muscle, or of the sciatic nerve, failed to produce contractions in the muscles in whose vicinity saponin had been injected. The same observation holds good of curarized animals. The ligation of the abdominal aorta did not prevent this paralysis.

Pelikan's results were confirmed by Köhler.§

Przybyszewski‖ says that the muscular fibres at the point of injection are greatly changed, the striations in many places become invisible, and the single fibres lose their regular form.

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*From this it may be inferred that this drug will prove a valuable therapeutic agent in those cases where it is necessary to produce an increased bronchial discharge.

†Under the title "skeleton muscles" all the striated muscles except the heart are here included.

‡*Abh. d. St. Petersb. Acad.*, 3, 15 Oct., 1867. *Gaz. Méd. de Paris*, 1867, No. 45.

§"Die lokale Anæsthesirung d. Saponin." Halle, 1872, Pfeffer.

‖*Arch. of Exp. Path.*, V., pp. 137, 142.

[The anatomical changes produced in the striated muscles after the application of saponin are apparently identical with those produced by a temperature of 36° to 40° C., or by post mortem rigidity.]

In a large number of experiments made by the author on frogs, sparrows, pigeons, guinea-pigs, cats and dogs, an injection of a solution* of saponin always produced paralysis of the muscle into which the injection was made. The painting of the muscle with a similar solution produced the same effect. The strongest faradaic currents failed to cause the poisoned muscles to contract.

As the experiments made all lead to the same conclusion, several only will be here given as illustrations:

Experiment made May 23, 1877. *Rana pipiens.* Exposed gastrocnemius of right side. Galvanical irritation with the current from two small Daniell cells, causes decided movement in the muscle at 11.22 A. M.

11.22½. The muscle was painted with a solution of saponin.

11.24. The same irritation applied to the painted muscle no longer causes it to contract.

11.25. Irritation with a current from the Daniell cells fails to produce contractions.

Experiment made May 15, 1877. *Rana temporaria.* Irritation (galvanic) with two small Daniell cells.

9.17. Irritation of left gastrocnemius causes the muscle to contract 4·6 millimetres, as measured by means of Marey's myographion.

9.17½. The left gastrocnemius was painted with the solution of saponin.

9.19. Irritation fails to cause the painted muscle to contract, though the other muscles of the animal contract when directly irritated.

In the preceding experiments on frogs the muscles *seemingly* lost their irritability to galvanic currents on having a solution of saponin externally applied to them; numerous other experiments proved that the same result was obtained—as was already noted by previous investigators—when the drug is injected into the muscle, in the different varieties of frogs, as well as in pigeons and mammals.

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*1 gramme to 10 cubic centimetres.

[In the paper as written by Dr. Lautenbach several experiments are usually given in proof of each statement, but wherever they agree closely we will only quote one type-experiment.—EDS.]

The same results were obtained when the faradaic* irritation was substituted for the galvanic, and consequently it would be a needless sacrifice of space and time to give examples of experiments where this method of irritation was employed.

To what is this paralysis due? Being purely local it may be due to one of several causes.

1. The drug may so disintegrate the muscular fibres as to prevent them from executing their functions. If this were cause of the paralysis, the greater portion of the muscular fibres would be found disintegrated after the application of saponin to them. In numerous microscopic examinations of muscles painted with this drug, and whose contractility had thereby been abolished, I indeed found a small number of fibres disintegrated, but only those to which saponin was immediately applied were so affected. In the experiments where the solution was painted on the muscles, only the more external fibres were disintegrated, those lying deeper escaping, and yet the muscles were paralyzed.

This last mentioned fact suggests another explanation for the paralysis following the local application of saponin, namely:

2. That the disintegrated muscular fibres act as a barrier to the passage of the electric current to the still healthy portion of the muscle, and consequently the latter does not contract. That such a loss of the receptive power of the muscle may occur and yet the muscle be still able to contract from impulses conveyed by its nerve, was proven by the author in a large number of experiments. If this were the explanation for the want of irritability in the muscle after the application of saponin, the painted muscle must of necessity contract when its nerve is irritated. In all the experiments where, from the application or injection of the drug, the muscle failed to respond to irritants locally applied, irritation of the nerves also failed to cause the poisoned muscles to contract; thus proving that the cause of the paralysis affected the whole muscle, and not only those portions which were disintegrated.

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*The apparatus used was a Leclanche cell with the Valentin modification of the DuBois-Reymond induction coil.

3. A drug may act as an irritant to the muscles, causing them to contract. Under these circumstances, the muscle being already contracted, a second irritation will have little or no effect, and the experimenter would be very likely to mistake the effects of a continued irritation for muscular paralysis.

Saponin is such a muscle-irritant. When applied to the gastrocnemius muscle, the latter very gradually contracts until its transverse is at least one-half the size of its longitudinal axis.

The following are illustrative experiments:

Time.	No. of Gal. Irritations.	Height of the Contractions.		REMARKS.
		From the Application of the Current.	From the Application of Saponin.	
Oct. 9, 1877.				*Rana esculenta.* After destruction of the brain and spinal cord the gastrocnemius of one extremity was removed and attached to Marey's myographion.
2. 15	1	10. 9 mm.		
2. 17	2	9. 5 "		
2. 18				The muscle was painted with a solution of Saponin.
			6. 4 mm.	
2. 19	3	2. 7 "		
2. 20	4	1. 8 "		
2. 20. 30			8. 9 "	
2. 21	5	0. 6 "		
2. 21. 30			9. 9 "	
2. 22	6	0. 4 "		
2. 22. 30			10. 1 "	
2. 24		0 "		
2. 25			10. 2 "	

In the above, as also in the following experiments, the painting of the muscle with saponin produced a gradually increasing contraction, independent of the action of any other irritant. The degree of this contraction when added to the degree of the contraction still produced by the galvanic irritation, shows the contraction resulting from the combined action of these two dissimilar irritants simultaneously applied. It is also seen that the effects of galvanic irritation are gradually abolished.

Time.	No. of Gal. Irritations.	Height of the Contractions.		REMARKS.
		From the Application of the Current.	From the Application of Saponin.	
Oct. 10, 1877.				*Rana esculenta.* In this experiment the sciatic nerve was galvanically irritated instead of the muscle.
3.06	1	5.6 mm.		
.08	2	3.5 "		
.08.30			8.3 mm.	Painted gastrocnemius with saponin.
.10	3	0. "	9.5 "	
.11	4	0. "	11.6 "	
.13	5	0. "	12.9 "	
.20	6	0. "	14.6 "	

In other experiments the galvanic or direct current was substituted by the induction or interrupted current. The induction apparatus used was that of DuBois-Reymond as modified by Valentin. The current employed was derived from a single Leclanche cell. Pflüger's modification of the Helmholtz myographion substituted that of Marey. The result was the same as in the preceding experiments.

These experiments show conclusively that muscles previously painted with saponin cease to contract, simply because the application of this drug irritates, and causes them to contract to their utmost, when, of course, no new irritation will cause any further contraction. Consequently such muscles are no longer irritable.

Saponin being a muscle-irritant, it behooves us to determine the nature of the irritation. Is this irritation neuro—or idio—muscular? In other words, does the drug irritate the nerves in the muscles or the muscular fibres themselves? An irritation of the nerves of a muscle produces *immediate* contraction of all the fibrillæ of that muscle. From the application of saponin the contraction is more gradual and occupies five, six, and even more minutes, in reaching its maximum. In this respect it approximates closely what Schiff has called the "*idio-muscular*" contractions."* If the contractions from saponin be of this variety, only the fibres directly involved by the drug will contract, while the remainder of the muscle will be quiescent. This contraction of the individual fibres will be manifested by their forming a protuberance over the level of the surrounding non-poisoned muscular fibres.

Such is indeed the effect seen when a pointed stick, previously dipped into a solution of saponin, is applied to a limited portion of a muscle, care being taken not to produce a mechanical irritation through the agency of the stick. Under these circumstances the part to which the drug has been applied is seen to gradually rise above the *niveau* of the surrounding fibres, and as gradually to return to its original position.

CONCLUSION.

Saponin directly applied to the skeleton muscles acts as an irritant, and produces an idio-muscular contraction. When this contraction has reached, or nearly reached, its maximum, the poisoned muscle ceases to respond to electrical irritations applied to the nerves or to the muscle itself.

Local action on the visceral muscles.—On the striated visceral muscles.—When saponin is directly applied to the heart of a frog the movements of this muscular viscus gradually become slower and less extensive, until they cease entirely.

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* *Lehrbuch d. Physiologie,* I., p. 21.

[When a weak solution of saponin is gradually injected into the heart of a frog, in whom the spinal cord and brain have been destroyed, it has a number of times been my fortune to find the skeleton muscles absolutely paralyzed as regards their neuro-muscular contractility. The muscles were by no means contracted by this injection ; the idio-muscular contractility remained. When the injection was pushed further the idio-muscular contraction occurred, and the muscles shortened and became much harder to the touch. I have been able to produce the same effect locally in mammals by injecting the solution very gradually into the femoral artery. In saponin we therefore have a substance which, when carefully applied, can destroy the activity of the nerves in the muscles, without affecting the proper or idio-muscular contractility.]

Oct. 12, 1877. Experiment on a frog, *R. temporaria.*

1.54. Exposed the heart. Heart-beats, 22 per minute.
2.03. Painted the heart with saponin.
2.06. Heart movements less marked.
2.10. 18 heart-beats per minute.
2.15. 15 " " " "
2.20. 4 " " " "

2.23. The heart's movements have ceased, and the muscle itself is no longer irritable. Reflex movements continue until 2.40.

July 2, 1877. Experiment on a frog, *R. pipiens.* Exposed the heart.

9.47. 31 heart-beats per minute.
9.49. Painted heart with saponin.
9.52. 24 heart-beats per minute.
9.55. 19 " " " "
10.01. 12 " " " "
10.04. 8 " " " "
10.10. 3 " " " "

10.12. The movements of the heart have ceased. The muscle itself is no longer irritable. Reflex movements continue.

The results obtained in the foregoing, and in a large number of similar experiments, might possibly be due to either a direct, or a reflex effect on the nerve centres. But as the same result was obtained in many experiments when both the brain and spinal cord have been previously destroyed, and even from the heart removed from the body, it must be concluded that this diminution of the heart's pulse, in frogs, is due to a direct action on its muscular fibres. When the central nervous system is destroyed, and saponin is injected into the *interior* of the heart, the same result is obtained.

In a manner similar to the above I have made numerous experiments on cats and rabbits. In rabbits the injection of .015 gram. and in cats the injection of .025 to .05 gram. of saponin into the heart, produced immediate cessation of the functions of that organ. That the electric current present in all muscles still persisted in these functionless hearts, was proved by the method which I first made use of in 1874 while engaged in determining the physiological properties of conium. This method consists in opening the pericardium, and then bringing the phrenic nerve in contact with the heart-muscle, when it will be observed that the diaphragm contracts. On taking the nerve from the heart, again frequently a second contraction occurs. When the hearts of mammals are paralyzed by the direct action of saponin, this phenomenon can still be produced.

These results justify the conclusion, that, *saponin locally applied to the heart, directly paralyzes this muscle.*

On the other striated visceral muscles, the *diaphragm*, saponin, locally applied, also acts paralytically, as injections into it always produce great interference with the inspiratory movements of mammals.

Action on the non-striated visceral muscles.—For obvious reasons my observations on this subject have not been very extensive. They have indeed been limited to the muscular coat of the intestines, and any one who has investigated the local action of drugs on these structures need not be told with what great uncertainty such experiments are conducted. The almost absolute impossibility of determining whether an apparent increased movement of the intestines *is* only apparent, is among the least of the drawbacks, for even if an increased peristalsis occurs after a drug has been painted or injected into the intestinal walls of an animal, it is still questionable whether this increased peristalsis was not due to causes other than those produced by the drug.

In my experiments: 1, the peristaltic action of portions of the intestines into the walls of which a saponin solution had been injected, or on which the solution had been painted, still occurred; 2, there was a very apparent increase in the peristaltic movements produced a short time after the application

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[When a weak solution of saponin is injected slowly into the heart of a frog, or rabbit, the heart very frequently ceases to beat in diastole. Should the injection be continued, an "arrest in systole," as Köhler calls it, *i. e.*, an idio-muscular contraction of the entire heart-muscle is produced.]

of the drug. This increased peristalsis may have been due to other causes than the drug.

Local action on the nerves.—The efferent nerves.—Pelikan and other observers have long since noticed that a solution of saponin applied directly to the nerves destroys the excitability of these tissues. My experiments have shown that this conclusion is true as far as it goes, but it does not go far enough. The increased excitability of the nerves which precedes the loss of function produced by saponin, has been observed by no previous experimenter, although on this subject numerous investigations have been made. The previous investigators failed to see this increased excitability because they made all their observations with the eye. Had the myographion been used this action would certainly have been observed.

In the earlier experiments I committed the same error as did Pelikan and Köhler, in not *measuring* the degree of excitability of the nerves, and consequently the conclusion was arrived at, that the sole action which the glucoside exerts on the efferent nerves is to diminish their excitability. Later, in the experiments made with the aid of the Marey and Pflüger myographions, different results were obtained.

The method resorted to in these latter experiments was to remove the gastrocnemius and sciatic nerve of a frog, and attach the former to the lever of the myographion. The nerve was placed on a cork and moistened with a 0.7 per cent. solution of chloride of sodium. The electrodes from a faradaic or a galvanic battery were applied to the nerve, each electrode being ten millimetres from its companion. A Valentin hammer was used to make and break the current.

[In some recent experiments the lower portion of the intestine of a frog recently killed was connected with a Ludwig's manometer. The intestine was filled with a solution of bicarbonate of sodium and tied at the stomach. A solution of saponin was now applied to the intestine, whereupon the mercury began to rise slowly in the manometer until the pressure had mounted to nearly thirty millimetres of mercury. Since this was a constant result, it can be concluded, that saponin irritates the unstriped muscles of the intestines as it does the striated heart and skeleton muscles.]

Time.	No. of Irritations.	Irritation.	Height of Contractions.	REMARKS.
Oct. 10, 1877.		Galvanic. 2 Small Daniell's.		*R. esculenta.*
1.45	1		2.2 mm.	
1.46	2		2.1 "	
1.47				Painted the portion of the nerve between the electrodes with saponin.
1.49	3		5.5 "	
1.52	4		1.7 "	
1.55	5		0. "	
Oct. 10, 1877.		Galvanic. 2 Small Daniell's.		*Rana temporaria.*
2.04	1		1.1 mm.	
2.05				Painted as in last experiment.
2.07	2		1.6 "	
2.10	3		0.3 "	
Oct. 9, 1877.		Induction Current. 2 in. covered.		*Rana temporaria.*
1.55	1		34.8 mm.	
1.56	2		34.9 "	
1.57½				Painted nerve as before.
1.58½	3		51.8 "	
2.00	4		64.4 "	
2.02½	5		64. "	
2.05½	6		55.8 "	
2.14	7		40.4 "	
2.18	8		32.3 "	
2.23	9		28.5 "	
2.31	10		9.7 "	By 2.30 the muscle ceased to contract when the nerve was irritated.

From the foregoing experiments it will be seen that the local application of a solution of saponin to an afferent nerve, produces an increased, followed by a diminished excitability, and, finally, by a total abolition of the functions of the portion of the nerve poisoned. The application of a solution of the drug to a nerve frequently produces contractions in the tributary muscles, showing that the drug acts on them as an irritant.

The primary increased excitability must be due to one of two causes: Either the nerve, when painted with the drug, becomes a better conductor for impressions received, or it offers less resistance to the reception of an irritation. If the former were the cause, irritation of a portion of the nerve situated to the central side of the part poisoned, would also produce an increased contraction of the muscle, after the lower portion of the nerve had been painted with saponin. This I have found not to be the case. The painting of the nerve with saponin did not, in the beginning, in the least influence the height of the contractions produced by irritation of the nerve above the part painted. The cause must, therefore, be sought in a decreased resistance offered to the reception of an irritation at the point to which the drug has been directly applied.*

The diminished excitability, which follows the primary augmented excitability, is due to a detrimental action on the conducting elements of the nerves themselves, as it is manifested not only at the point of application, but also in all portions centrally situated from this point.

CONCLUSION.

Saponin directly applied to an efferent nerve, first excites and then paralyzes it. The excitation is due to an increased receptive power (i. e., a decreased resistance to the reception of the irritation) at the point of application. The secondary paralysis is due to an interference with the functions of the conducting elements of the nerve.

On the afferent nerves.—Experiments on *frogs.* When a solution of the drug is directly applied to the sciatic nerve of

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*The difference between the receiving and conducting properties of nerves was first alluded to by Schiff (*Lehrb. d. Phys.*, I., 1859), and has since been proven by the author.

a frog, it first of all increases the reflex excitability of the part on which it is painted. This increased excitability was proven by applying to a "saponized" nerve an electric irritation, which was previously not strong enough to produce reflex contractions in the opposite limb; while after the application of the drug, the same current was found to produce marked reflex contractions. As with the efferent nerves, this increased excitability is soon followed by the reverse effect; and in a very short time strong electrical irritations fail to produce any reflex contractions whatsoever. The sensory nerves of frogs are, therefore, first excited and then paralyzed by saponin.

On the afferent nerves of *mammals* the results obtained will prove of great interest to physiologists, who in saponin will find an important agent to determine the presence of afferent nerves in organs where their presence has long seemed doubtful. When a solution of the glucoside is injected into an efferent blood-vessel, a rise in the blood pressure is observed. As will be shown later, this rise is caused by a stimulation of the vaso-motor "centres." When, however, the afferent nerves of a limb or organ into which the drug is injected have previously been cut, this increased blood pressure fails to occur, and, consequently, must be due to a direct irritant action of the drug on these afferent nerves, causing a reflex stimulation of the vaso-motor "centres."

Saponin is the only substance at present known to have such an action.

To determine whether an organ possesses afferent nerves or not, it is only necessary to inject a solution of the glucoside into the artery of the organ in doubt; and if the arterial pressure immediately rises, that alone will be presumptive evidence of the presence of sensory nerves. If, however, the nervous communications with this organ are now cut, and it is found that a rise in the pressure no longer follows the injection of the drug, it is proven, beyond a doubt, that the organ contains afferent nerve fibres.

I have proven in this manner the presence of nerve fibres in the brain, which are afferent to the vaso-motor "centres" in the medulla oblongata. An injection of saponin into the carotid artery (internal), produces an immediate rise in the arterial pressure. This might be due to a direct action on the vaso-motor "centres" themselves; but as it failed to occur after section of the pons, though the poison still reached these "centres," the conclusion was inevitable that by this section I destroyed fibres which carried the impression made by saponin to these "centres." These experiments led to another very interesting observation, namely, the course which the vaso-motor fibres take after they leave the medulla oblongata. In two experiments, one on a rabbit and the other on a dog, I cut the whole of the posterior four-fifths of the cervical spinal cord, immediately below the calamus. An injection of saponin was then made into the internal carotid artery, when an immediate increase in the arterial pressure occurred, showing conclusively that the vaso-motor fibres of the spinal cord had not been cut. An injection into the femoral artery in these animals had no immediate effect on the blood pressure; and, consequently, the sensory fibres of the cord must have been cut. The vaso-motor fibres must at this point pass through the anterior fifth of the cord.

The method employed appears so certain, that physiologists will, no doubt, adopt it to further determine the course of these nerves.

The only other organ which I have thus far searched for afferent nerves, is the liver. The efferent vessel used in these experiments, was the vena porta. An injection of saponin into this vein, produced an immediate augmentation in the arterial pressure. Concluding from this, that afferent nerves existed in this organ, the question, Which are the afferent nerves? presented itself. It seemed to me most plausible that these nerves were branches of the pneumogastrics; and I was, therefore, much surprised to find that, after section of both pneumogastrics in the neck, an injection of saponin into the liver still augmented the blood pressure.

It was, therefore, necessary to examine the other nerves which form the hepatic plexus for the afferent nerves of the liver. The only ones given by anatomists are branches from the two upper splanchnics, and from the phrenic nerves. Section of the latter I found not to influence the effect of the injection. The splanchnics could only be studied by exclusion, as they are too intimately connected with the larger number of the blood-vessels of the body, to admit of their section without vitiating the results. The splanchnics receiving filaments from the spinal cord as low down as the eleventh dorsal vertebra, a section of the cord was made one vertebra below this. On injecting saponin into the liver of an animal thus prepared, no increased arterial pressure was observed. As an injection of the glucoside into one of the thoracic arteries still affected the blood pressure, it must be concluded that the afferent nerves of the liver are not branches of the pneumogastric, splanchnic, or phrenic nerves; but are hitherto undescribed nerves, entering the spinal cord below the twelfth dorsal vertebra.

There are other evidences of an excited neurility of the afferent nerves following the introduction of the drug into an efferent (from the heart) blood-vessel. Thus, for instance, an injection of .15 gram. of this substance into the femoral artery of a cat or rabbit, produces convulsions, which, for reasons to be given shortly, I ascribe to the direct stimulation of sensory nerves.

Convulsions may be produced by a drug in four different ways: First—they may be cerebral; second—they may be spinal; third—they may be due to stimulation of the peripheral ends of the motor nerves; fourth—they may be caused reflexly by stimulation of the peripheral ends of the sensory nerves.

That the convulsions are not due to a direct stimulation of the peripheral motor nerves was readily proved by tying the iliac artery of one side in various animals and then injecting saponin peripherally into the femoral artery of the opposite side, when convulsions uniformly occurred over the whole body. Were they due to a direct action on the peripheral efferent nerves they would not have occurred in the limb where the access of the poison to the nerves was cut off.

Dec. 18, 1877. Experiment on a cat. Tied the right external iliac artery; placed the needle of the hypodermic syringe into the left femoral artery. 2:40 P. M.—Injected .15 gram. saponin into the left femoral artery, towards the periphery. 2:40:30 P. M.—Clonic convulsions, involving the whole of the muscles of the extremities, including those of the right posterior extremity. These were followed by opisthotonos and tetanus, involving all four extremities. The animal recovered.

Dec. 27, 1877. On a rabbit. Tied right external iliac artery. Placed hypodermic needle in a branch of the left femoral artery — the needle pointing towards the periphery. 11:17 A. M.—Injected .1 gram. of the drug into this prepared artery. Immediate clonic convulsions were produced all over the body. The animal recovered.

Eight of these experiments were made, all giving similar results.

I was forced to conclude that these convulsions were due, not to a direct effect on the brain or spinal cord, but to stimulation of the peripheral sensory nerves, reflexly exciting the spasm-producing mechanisms in the spinal-cord or brain, for the following reasons:

1. Convulsions do not occur when the drug is injected into a vein of the general circulation. Were they due to a direct effect on the nerve centres they would occur even more readily if the drug was introduced into a vein, when it could the more easily reach the centres. It is true that convulsions frequently follow the subcutaneous introduction of the drug in mice ; but as these occurred immediately after the injection, and it taking hours for the drug to be absorbed under these circumstances, these experiments only go to prove the correctness of my statement. These convulsions could only have been due to a direct action on the afferent nerves.

2. Convulsions were produced in cats and rabbits, in whom I tied the abdominal aorta and inferior *vena cava*, and then injected the drug into the femoral artery of one of the posterior extremities.

3. In a cat whose heart was paralyzed by chloroform, and in whom the circulation had ceased, an injection of .15 gram. into the left femoral artery, after previously tying the corresponding femoral vein, produced tetanic convulsions in all four extremities. In spite of the cessation of the circulation, this animal evidently had enough of the central nervous system intact, to translate the impression received from the afferent

nerves of the injected limb into tetanus-producing efferent impulses.

4. When the nervous communications between the centres and the limb, into whose efferent vessels saponin is injected, are destroyed, convulsions will not only fail to occur in that limb, but will also fail to be present in any other portion of the body. Thus, for example, in animals where I cut the anterior crural and sciatic nerves of an extremity, and then injected a solution of the drug into an artery of the same extremity, no convulsions were produced, nor were any symptoms manifest for hours. If, on the other hand, the nerves were allowed to remain intact, convulsions occurred immediately on the injection of the drug. In the same manner, an injection into the vena porta will produce *immediate* convulsions, extending over the whole of the body; and as these no longer occur anywhere when the spinal cord is cut at the twelfth dorsal vertebra, it must be conceded that they are due to a direct action on the afferent nerves of the liver.

That the brain contains nerve fibres which are afferent to the vaso-motor "centres" was demonstrated on one of the previous pages. Either these fibres or others contained in the brain are afferent to the spasm-producing mechanisms of the central nervous system. This was shown in my experiments, by the occurrence of convulsions immediately on introducing the drug into the internal carotid artery, and their non-production by such an injection after section of the pons immediately below the corpora quadrigemini, when, evidently, the connection of the fibres, stimulated by the drug with the so-called spasm-centre, or centres, was destroyed. After this operation, however, convulsions still result from an injection into the femoral artery.

Having shown that this symptom of saponin-poisoning is due to a direct stimulation of the peripheral afferent nerves, it remains still to be determined whether the stimulation

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[When the entire central nervous system of a frog is destroyed, and a very strong solution of saponin is injected through the heart into the abdominal aorta, tetanus of the posterior extremities occurs. This is probably due to a direct action on the nerves. This tetanus is in a short time replaced by the idio-muscular contraction of the muscles.]

reflexly excites the spasm-"centres" of the brain or those of the spinal cord.

For the following reasons it was concluded that the former and not the latter are reflexly excited:

1. When, through the introduction of saponin into the femoral artery, convulsions were produced, these immediately ceased on making a section of the medulla oblongata at the calamus; nor could they be again brought on by fresh injections of the drug. Were they due to a reflex excitation of the spinal spasm-"centres" they must have continued even after the section; but as they ceased immediately on breaking the connection of the brain with the nerves affected by the drug, the inevitable conclusion is that the structures reflexly affected in producing these convulsions must be in the brain.

2. When an injection of the drug is made into the femoral artery after section of the medulla oblongata at the calamus, no convulsions occur.

The conclusions to be drawn from the above given facts are, that *the saponin convulsions are due to a direct stimulation of the peripheral afferent nerves, reflexly exciting the convulsion-producing mechanisms in the brain.*

Thus far the convulsion-producing mechanism has been limited, by the experiments above referred to, to the portion of the central nervous system limited by the corpora-quadrigemina above and the calamus below. To determine whether or not this "centre" is located in the medulla oblongata, I, in a number of experiments on cats, cut this organ at its junction with the pons. On now introducing the drug into an efferent blood-vessel of one of the extremities, no convulsions were produced, showing that the spasm-"centres" reflexly affected by saponin are situated somewhere in the pons, and not in the medulla oblongata. This corresponds to the observations of Nothnagel,* who also locates these "centres" in the pons.

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* *Virchow's Archiv*, B. 44, H. 1, p. 1.

[Since the above was written, Luchsinger has shown, that certain precautions are required to demonstrate the action of convulsive agents on the spasm-centres situated in the spinal cord (*Pflüger's Archiv*).]

CONCLUSIONS.

Directly applied to the afferent nerves of frogs, saponin first acts as an exciting, and then as a paralytic agent.

The afferent nerves of mammals are excited by the direct action of the glucoside, producing reflexly an augmented blood pressure and convulsions.

The increased blood pressure is due to the excitation of the afferent nerves, reflexly producing a stimulation of the vaso-motor mechanism in the medulla oblongata.

The convulsions are due to the same excitation reflexly stimulating a spasm-producing mechanism in the pons.

Action on the spinal cord.—Köhler* found that when the brain of a frog is destroyed, and a saponin solution is directly applied to the spinal cord, convulsions result. In the author's experiments the same effect was observed, but it was soon followed by spinal paralysis and complete abolition of reflex activity. Convulsions were never observed from this local action on the cord, so long as the brain remained intact.

The following are illustrative experiments:

Dec. 19, 1877. Experiment on a frog, *R. esculenta.* Exposed the spinal cord below the origin of the nerves of the anterior extremities. Painted the exposed portion of the cord with a solution of saponin. In two minutes, irritation of a posterior extremity causes reflex movements in it and its fellow, but not in the anterior extremities. Irritation of an anterior extremity causes reflex movements in the anterior portion of the animal, but not in the posterior portion. This shows that the conducting power of the spinal cord has been lost between the origin of the anterior and posterior nerves. In five minutes convulsive twitchings were manifest in the toes of the posterior extremities. At the end of seven minutes these twitchings ceased, and reflex movements could no longer be excited, though they still occurred in the anterior extremities.

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*"Die lokale Anæsthesirung d. Saponin," Halle, 1872.

[But if saponin is directly injected into the medulla oblongata, a tetanus lasting a few minutes only, is immediately produced, after which it is absolutely impossible to bring about any reflexes in the frog, although the motor nerves are still excitable.]

July 30, 1877. Experiment on a frog, *R. esculenta.* The spinal cord was painted with saponin, the brain being left undisturbed. In five minutes complete paralysis, both reflex and voluntary, of the posterior extremities. No convulsions. In ten minutes the convulsions had not appeared.

Action on the brain.—The brain of the *frog* being so subordinate a portion of the nervous system, as compared to the spinal cord in this animal, I was not surprised to find the application of saponin to their brains produce no *apparent* symptoms. This absence of symptoms was, however, only apparent, as on closer examination it was found that the tactile sense* was lost, and that the tri-facial reflexes no longer occurred. The frogs whose brains had been thus treated, lost their sense of sight, and perhaps this will account for the backward movements of such animals. Reflex movements continued to occur. Convulsions were never observed. The animals usually recovered in five or six hours.

On pigeons similar effects were observed. After painting their hemispheres with a solution of the glucoside, these animals fell into a sleep, from which they wakened after some hours. From this state the animals could be aroused by strong irritants, but when the irritant was removed the symptom returned. The same backward movements were observed as in frogs.

On mammals.—In these animals various portions of the hemispherical convolutions were painted with saponin, when, for the time being, the functions of these portions of the cerebral lobes seemed to be entirely abrogated. For example, when the convolutions in the neighborhood of the sulcus cruciatus were painted, the peculiar paralysis of tactile sensibility was observed. These symptoms afterwards disappeared. From this it appears that a solution of *saponin directly applied to the cerebrum temporarily destroys the functions of the part involved.*

The injection of a few drops of the solution into the corpus lenticularis* yielded interesting results. It has long been an object among physiologists to determine the cause of hemiplegia from cerebral lesions. Saponin is evidently a substance which will greatly assist in the accomplishment of this object. The explanation of this hemiplegia most universally adopted at present, is, that it is due to the destruction of certain portions of the brain, whose function is to preside over the movements of the opposite side of the body. This explanation would be quite plausible were it not that these portions of the brain have frequently been found entirely disintegrated in persons who never were hemiplegic. In dogs I have several times made a complete section of the brain on a level with the upper boundary of the corpora quadrigemina, without motor paralysis resulting. In a number of other experiments the whole of the corpus striatum and optic thalamus of one side were destroyed without ever producing hemiplegia. Cerebral hemiplegia not being due to the destruction of any of the great ganglia of the brain, it is somewhat difficult to determine why a hemorrhage into these structures so frequently produces hemiplegia. Apoplexy may possibly produce this symptom in three different ways: first, it may destroy the voluntary motion-producing nervous mechanism; second, it may through pressure influence this mechanism; third, it may irritate fibres concerned in motion, and thereby produce paralysis.

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*Frogs always lose their tactile sense when the cerebrum is removed.

An injection of saponin into the corpus lenticularis produced hemiplegia of the opposite side of the body. That this effect was not due to pressure, or to the destruction of the part, was proved by experiments where water was injected into this portion of the nervous system without producing any paralysis. This hemiplegia from saponin is but temporary, it vanishing after some hours, and it was only observed in three experiments; but as this is the first instance on record† in which hemiplegia was traumatically produced in animals, it is well worth dwelling on.

Saponin must produce this effect through its irritative properties, and as we have before seen that it is a local irritant to the peripheral nerve fibres, it is by no means improbable that it similarly affects the fibres in the brain. What at first sight appears improbable, is that an irritant applied to a portion of the nervous system can produce motor paralysis, an excitation of motor fibres being usually associated with motion. A number of other facts go to confirm the apparently improbable statement, that an irritant applied to nerve fibres may prevent these from exercising their functions. A well known fact, to which the writer has, within a short time, again drawn attention, is that a polarized* nerve, under certain conditions, fails to respond to irritants applied to it *anywhere*† throughout its course. Undoubtedly the pressure of saponin in the corpus lenticularis acts on fibres concerned in producing motion, as does the constant current on the ordinary nerves; and the consequent absence of movement in certain muscles is due to the polarization preventing the transmission of the voluntary motion-producing impressions.

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*The posterior portion of the corpus striatum.

†I have on other occasions, and by other agents, succeeded in producing hemiplegia in dogs and cats, and will in a later paper draw attention to these experiments.

Local action on blood, lymph, mucus and pus corpuscles.—The only previous investigation of the action of saponin on the blood-corpuscles was made by Przybyszewski,‡ and his experiments do not seem to have yielded any very positive results. This author found, that if a drop of a four per cent. solution of the drug was mixed with a drop of frog's blood, the stroma of the red corpuscles becomes pale, until only a granular nucleus remains visible—this latter only differing from the white corpuscles in being smaller and more oval. In his experiments on human blood, with a ten per cent. solution, he found (p. 140,) "in 3–5 minutes the upper surface of the cells to seem folded and contracted, and soon the red corpuscle becomes so pale that its stroma resembles a granular residue. In a few minutes the whole preparation was filled with irregularly-shaped pale corpuscles."

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*A polarized nerve is one through which a constant or polarizing current is passing.

†Pflüger has denied this, but I have repeatedly demonstrated it to the satisfaction of several eminent physiologists, who were at first indisposed to believe it.

‡*Arch. f. exp. Path.*, V., pp. 137–142.

No doubt the preparation of the glucoside used by this experimenter was not all that could be wished, or he would have made the interesting discoveries in regard to this action of the substance which have fallen to the lot of the author. It may however be alleged, that the author's preparation of saponin was impure, and that the phenomena hereafter to be described were due to the specific action of the impurities. This objection might be plausible: first, if we knew of any other neutral substance which had a similar action on the blood; second, if the experiments had been made with but one specimen of the drug. Saponin from the Brazilian soap-bark, from the American and Oriental soapworts, and from senega snake-root, all gave identical results. The water which was allowed to percolate through powdered soapwort, soap-bark, or senega snake-root, was found to have the same action on the blood as saponin.

In studying this action, the lower classes of vertebrates will be taken first.

Action on the blood of *frogs.*—When a drop of saponin solution (1 gram. to 10 c. c.) is allowed to mingle with a drop of frog's blood, as with a flash the color of the red corpuscles becomes very faint, but every portion of these corpuscles remains visible. It appears as if the coloring matter of the corpuscles is dissolved, leaving behind only the frame-work. The granular nucleus of these corpuscles remains unaffected, and, on superficial examination, appears to be a lymph corpuscle, which it strongly resembles. Sometimes the frame-work of globulin also undergoes changes, and crystallizes, the crystals being elongated rhombs, and *colorless.** Much slower than the red, the white corpuscles are affected, and seem absolutely to disappear.

The cause of the red corpuscles becoming almost invisible, is the dissolving out of the coloring matter, making the refracting angle of these corpuscles nearly identical with the refracting angle of the liquor sanguinis. That such was the case, I proved by adding a trace of tr. iodinii to frog's blood, previously treated with saponin, when almost instantaneously the red corpuscles again became very apparent; that is, the almost absolute invisibility of the red corpuscles is overcome by placing around it a fluid of a different refracting power.* The invisibility of the white corpuscles continued, in spite of the iodine, making it very probable that these were dissolved. The lymph corpuscles were not affected by either the saponin or the iodine solution.

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*These crystals are in shape identical with the so-called hæmoglobin crystals, differing from these only in not containing any coloring matter. This, taken in connection with the fact that saponin can dissolve out the coloring matter of the hæmoglobin and allow the transparent colorless crystal to remain, makes it certain that the formation of blood-crystals may be entirely independent of the coloring matter, and that the latter, when present in them, must be looked upon as a chance production, as iron is in a quartz crystal.

On the blood of *pigeons.*—On applying a drop of the solution of the drug to the blood of these animals, and then examining the latter under the microscope, it was found that the red corpuscles — nucleus and all — had completely disappeared. In a few minutes the white corpuscles were also found to have disappeared. This vanishing of all the corpuscles must have been due to their being dissolved, as iodine failed to make them again appear.

On mammals.—First the red and then the white corpuscles disappear, on adding saponin to blood taken from mice, guinea-pigs, cats, dogs and men. On taking a drop of human blood, and placing it on a glass slide, and over it a cover-glass, and then placing at the edge of the latter a drop of the solution of the drug, the red corpuscles were seen to disappear immediately, while the white usually occupied at least five or six minutes before they too were dissolved. I say dissolved, because after such an experiment the tincture of iodine method failed to demonstrate the presence of morphological bodies. At a later period, frequently the microscopic slide was found to be covered with numerous transparent, colorless "hæmoglobin" crystals.

If a large quantity of blood, taken from a mammal, is mixed with a small quantity of saponin solution, and then allowed to stand, the blood will not coagulate for five or eight days. This great retardation in the coagulation of the blood treated with this drug, I find at present impossible to explain; but it is an interesting fact, and well worth recording. Blood treated in this manner, shows most beautifully the spectrum for oxy-hæmoglobin.

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* The microscope employed was a Seyss; objective number, 5; ocular number, 1.

These experiments were made in the months of January and December.

The "saponized" corpuscles do not reappear on adding iodine or carmine * to the preparation, and, consequently, their disappearance is not due to their refraction being made identical, or nearly so, with that of the blood plasma, through the hæmatin being dissolved away from the globin. Their disappearance is not due to the imbibition of water, as a drop of water placed on a slide, on which corpuscles have been prepared, is insufficient to make them indistinct.† It can also not be due to an inorganic chemical solvent in saponin, this substance containing only oxygen, hydrogen and carbon, and being of a neutral reaction. The most careful chemical examination failed to detect traces of any of the alkalies. From these facts it must be concluded that the glucoside is a solvent for the red as well as the white corpuscles of mammals.

Having come to this conclusion, it was necessary to determine what percentage of the glucoside was necessary in order for the solution to have its peculiar effect on the corpuscles. It was found that sometimes, but not always, a drop of a solution of saponin, of the strength of one part to two thousand parts of distilled water, would cause the solution of the corpuscles in three times its bulk of blood. A solution of the strength of one part to one thousand of distilled water constantly produced this effect.

When a microscopic slide on which is placed non-saponized blood, is slightly heated, *debris* of corpuscles immediately appear. If, however, "saponized" blood is so treated, the *debris* does not appear until the preparation is almost dry. Sometimes, on adding the drug to blood, *debris* of corpuscles appear, which may or may not, according to the prejudices of the observer, be considered as parts of corpuscular walls.

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* Gaucher (*Arch. de Phys.*, IV., p. 770) found that when he dissolved away the coloring matter of red corpuscles, these could readily be stained with carmine.

† Water, in large excess, causes them to disappear.

When a large amount of the drug is introduced into an efferent* blood-vessel, such as the femoral artery or mesenteric vein, the corpuscular elements of the blood gradually become less numerous. This is more especially true of the red corpuscles, and we have, consequently, produced a pseudo-leucocythæmia, i. e., the colorless corpuscles are not more numerous than before, but through the solution of some of the colored corpuscles, they are, relative to these latter, increased in numbers.

Saponin being a solvent for colorless corpuscles, is it also a solvent for mucus and pus corpuscles? Without having made the necessary experiments, the inclination would be to answer this question in the affirmative, as the histologists teach us that these latter are colorless corpuscles which have migrated through the walls of the blood-vessels. Many histologists go so far as to say they are identical with the colorless corpuscles. No proof has, as yet, been furnished of the migrated colorless corpuscles being the pus and mucus corpuscles; though Waller, Cohnheim and others, have shown that the former do migrate; but that they become the latter remains still to be proven. The statement that these corpuscles are one and the same, rests solely on assumption, as they are not even morphologically identical in all respects.

Should the mucus, pus and colorless corpuscles of the blood be identical, it would be expected that they would all three be soluble in a saponin solution. When mucus is taken from the nose, mouth or œsophagus of man and other mammals, and treated with this solution, no effect is observed. After continuing the observations for several days, the mucus corpuscles were always found to remain unaltered. Pus taken from abscesses was treated in the same way, without any of the pus corpuscles being dissolved. It must, therefore, be concluded that pus and mucus corpuscles are not identical with the colorless corpuscles of the blood.

Another question which here suggests itself, is, whether or not these latter are identical with the lymph corpuscles from which they are said to originate. Lymph corpuscles obtained from the thoracic duct and the lymphatic glands of mammals, or from the lymph sacs of frogs, are not in the least affected by saponin. So that the last query must also be answered in the negative.

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* The drug could not be introduced in large quantities into a vein of the general circulation, for fear of producing the direct cardiac action.

For fear that some mistake might underlie these experiments, they were repeated scores of times with saponin obtained from different sources, but the same results were always obtained.

CONCLUSIONS.

1. *Saponin dissolves out the coloring matter from the stroma of the red corpuscles of frogs.*
2. *It is a solvent for the whole of both red and colorless corpuscles of birds and mammals.*
3. *It is not a solvent for either the lymph, mucus, or pus corpuscles.*
4. *The white or colorless corpuscle of the blood is not identical with the lymph, mucus or pus corpuscle.*

A question of great clinical and pathological importance here suggests itself, namely, whether the proliferated "white corpuscles" in leucocythæmia can be dissolved by a solution of saponin, as well as the white corpuscles in normal blood. By determining this, we can, perhaps, come to some result which will enable us to diagnose whether in a given case of this disease the increased number of leucocytes is not due to the presence in the blood of numerous lymph or other corpuscles not identical with the white blood corpuscles. My opportunities to pursue the subject in this direction have been few indeed; but I hope the subject will be taken up by physicians who have such cases under observation. In but one case of true leucocythæmia has this method of examination been resorted to; this was in the case of an old man, much emaciated, and in whom the spleen was somewhat enlarged. The white corpuscles in his blood appeared to be increased to at least twenty times the normal number. On adding saponin to a drop of his blood, the white corpuscles disappeared, but a large number of granular corpuscles, some of the size of lymph corpuscles, and others smaller, made their appearance. These may have been nuclei of the white corpuscles. In several places in the field under the microscope, granular nucleated cells, larger than white corpuscles, were observed.

The blood in a case of pseudo-leucocythæmia was affected by the drug in the same manner as normal blood.

[Saponin immediately arrests the movements of spermatozoa and other ciliated cells. It destroys at once all infusorial life with which it comes in contact, but fails to influence in the least degree the movements of vibrios and bacteria.]

[*To be Continued.*]

ART. IV.—RETROGRADE AND LATERAL MOVEMENTS WITH HYPNOTISM.

BY ISAAC OTT, M. D.

WHEN cold is applied to certain definite regions of the skin in pigeons, they exhibit retrograde movements alternating with fits of stupor. The agent, so far, has been rhigolene, to produce the necessary cold. Ether, when vaporized, was totally unable to produce any effect. The region to which the cold must be applied is the skin of the back of the neck. These phenomena were first observed by Dr. S. Weir Mitchell. I have lately been studying the effect of irritants on the skin of pigeons, and the seat of the phenomena produced. I found that ether, alcohol, chloroform and nitrite of amyl were powerless to produce the effects seen after the application of rhigolene. If, however, bisulphide of carbon was dropped on the skin of the back of the neck, then all the phenomena produced by rhigolene ensued in a marked manner. Thus a single drop of the bisulphide of carbon applied to the back of the neck of the pigeon caused him to retrograde and to pass into states of quietude. When bisulphide of carbon is applied to the skin of the neck in pigeons, the birds run forward as if no agent was acting on them, but suddenly they commence to run backwards, it being quite evidently against their will, as they seek to overcome the tendency. During the period of quietude, the body sinks down somewhat, the head being drawn in close to the body and bent towards the ground to a considerable extent. That these phenomena are due to a simple irritation of the nerves, is proved by an experiment where I attached a small bull-dog forceps to the skin on the back of the neck, when similar retrograde movements ensued, followed by a period of quietude. As remarked by Dr. Mitchell, these phenomena are reflex in their nature. Here the mechanical irritation of the sensory nerves is reflected on the central nervous system, causing it to evolve the phenomena under consideration. To more accurately determine the seat of these phenomena, I have made some experiments: In a pigeon under ether, the skull was trephined and the cerebellum broken up. When the bird recovered from the operation, that is several hours afterwards, rhigolene was applied and the bird exhibited the same phenomena as an uninjured one. In another pigeon the skull was trephined and the cerebrum broken up; then, after a period of several hours, the bisulphide of carbon was dropped on the skin of the neck, when the pigeon began to make retrograde movements and to have periods of quietude. The bird did not run forward, which was due normally to cerebral action when the bisulphide was applied. Hence, the forward movement after the application of either rhigolene or bisulphide of carbon, was due to cerebral activity. These series of experiments seemed to demonstrate that the cervical reflex had its central origin at the base of the encephalon, for the cerebrum and cerebellum did not seem necessary for the production of the phenomena in question. Section of the semicircular canals had no effect on these phenomena, except to make them more complicated.

Now, when the cerebrum is destroyed, the nervous system of the bird is a mere automaton, played upon by appropriate external agents. These agents, in the phenomena under consideration, are rhigolene, bisulphide of carbon and mechanical irritation. The inquiry now arises: how do you explain the phenomena in question? It is well known, since the time of Magendie, that in the corpora striata are seated ganglia, causing the animal to run backward Not only does the bisulphide, when placed on both sides of the median line of the back part of the neck, cause the bird to run backward, but when it is placed to the right of the median line, the bird runs in a circle towards the left. A drop on the left of the median line causes the bird to run to the right. The rule here is, that the bird makes a circular movement opposite the irritated side. It has seemed to me that these phenomena of lateral movements in pigeons can be explained as follows: According to Chauveau, the sensory fibres do not decussate in pigeons, and if an irritation is made on one side of the median line the impression ascends on the same side, and calls the retrogressive ganglia of that side only into activity, which activity is expressed on the opposite side of the body, due to the motor decussation. Now the retrogressive ganglia are in power over one side of the brain, whilst the other side of the brain, under the influence of the cerebrum, is disposed to move the opposite side of the body forward; hence the bird cannot go directly forward, but deviates to the side where the retrogressive ganglia have shown their activity—the bird moves in a circle. The action might be compared to that of driving a horse forward, and at the same time strongly pulling one of the reins, the checking rein corresponding to the side of the brain under the influence of the retrogressive ganglia. If now the skin on both sides of the median line is irritated, then the retrogressive ganglia on both sides of the brain are dominant, and the bird moves backward, notwithstanding all efforts to prevent it. It might be compared to a horse pulled on his haunches by two strong check-reins. The periods of quietude, either preceding or following the external application of bisulphide of carbon, are to be explained. It is well known by Kircher's "experimentum mirabile," that chickens, when held down, pass into a state, called by Czermak, hypnotic. In fact, Czermak has been able to produce a hypnotic state in pigeons and other birds. He found if he held the pigeon on his back and rubbed him softly in the parotid region, that the pigeon closed and opened his eyes, made strong respiratory movements and passed into a state of perfect quietude, called by him hypnotic. In my experiments on pigeons, the state of quietude is actually hypnotic, as the bird exhibits similar phenomena, opens and closes his eyes, breathes heavily, and remains perfectly quiet. It strikes me that this hypnotic state can be explained by sensory irritation, produced by the bisulphide of carbon calling ganglia at the base of the brain into activity, which ganglia have an inhibitory power. That sensory irritation may come into play is shown by Levissons' experiment with the frog, where simply tying his anterior extremities and placing him on his back keeps him in a state of quietude. The rapid breathing also indicates a strong sensory irritation. In my experiments, the temporary irritation explains the temporary hypnotism. With this method of viewing matters, the phenomena of hypnotism have nothing to do with the cerebrum, but are caused by ganglia at the base of the brain inhibiting the will.

In cats and rabbits the application of bisulphide of carbon to the skin on the back of the neck, causes them to run forward and to leap up in the air. I have not been able in animals, so far, to produce movements similar to those seen in pigeons, but hope shortly to find some animal in which they can be produced. This whole subject is important, not only to the physiologist, but also to the pathologist.

ART. V.—THE TENDON-REFLEX: ITS PREVALENCE AND ITS PHYSIOLOGY.

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IN 1874 Professors Erb and Westphal each published in the same number of the same journal* a paper on what has come to be known in English as the "tendon-reflex," frankly sharing the honors of priority with each other, and thus presenting an example of magnanimity too rarely emulated by professional men. They were the first to bring into notice the fact that when the tendon of the quadriceps extensor muscle of the leg was lightly and quickly struck below the knee, there immediately followed a jerk forward of the foot and leg. As it is almost superfluous to remark by way of reminder, they attached great value to this phenomenon as a symptom of disease, since Westphal claimed that its absence was pathognomonic of locomotor ataxia, while Erb asserted that its presence in an exaggerated degree was constant in spasmodic spinal paralysis. The literature of the subject from that period to the present date would fill several portly folios. Schultze and Fürbringer,† Berger,‡ Henze,‖ Sachs,§ Burckhardt,¶ Lewinski,** Westphal again,†† Tschirjew,‡‡ Muhr,‖‖ Gowers,§§ Sawyer,¶¶ Hamilton,*** Bannister †††; all have

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**Arch. f. Psych.*, 1874. S. 792, 803.

† *Centralbl. f. d. med. Wissensch.*, 1875, No. 54.

‡ *Sitzb. d. Schles. Gesellsch. f. vaterländ. Cultur.*—Med. Sect., 23 Juli, 1875.

‖ *St. Petersb. med. Wochensch.*, 30 October, 1876.

§ "Die Nerven der Sehnen," *Reichert u. DuBois-Reymond's Arch.*, 1875. S. 402.

¶ *Festschr. d. Andenken an A. v. Haller*, Bern, 1877.

** *Arch. f. Psych.*, VII. S. 327, 1877.

†† Ibid., VIII. Hft. 2, and *Berl. klin. Wochenschr.*, No. 1, 1878.

‡‡ *Arch. f. Psych.*, VIII. Hft. 3.

‖‖ *Psych. Centrbltt.*, No. 2, 1878.

§§ *Brit. Med. Journ.*, Aug. 31, 1878.

¶¶ Ibid.

*** *Bost. Med. and Surg. Journ.*, Dec. 19, 1878.

††† This journal, October, 1878.

contributed, in varying degree, to the question. It cannot be gainsaid that the clinical importance of the phenomenon has been eagerly appreciated in this country and abroad; and whatever tends to its elucidation, be it ever so slight, or to a more exact comprehension of its practical applicability, as well as of its relations to the recognized mechanisms of the cerebro-spinal axis, should not only possess an intrinsic interest but also an adventitious one lent to it by the present current of neurological thought.

It has seemed to me that the significance of the tendon-reflex in disease is so largely dependent upon exact knowledge of its modes of existence in health as to warrant an investigation into the latter. I have, therefore, endeavored to obtain some guiding facts in regard to its normal prevalence in the adult and the child, its usual site, the variations in the degree of its manifestation, its relative strength upon the two sides. In the course of this inquiry, I have, for the sake of uniformity, observed certain invariable rules. I have always made use of the tendinous expansion below the patella of the quadriceps extensor muscle. I have always taken care that the thigh should be supported by my arm placed in the bend of the knee, the leg dangling passively. I have always tapped upon the bare skin; and, finally, I have selected subjects about whose health, past and present, there could be no suspicion.

Prof. Westphal makes the positive statement in his latest article that he has yet to find a person in whom the tendon-reflex of the quadriceps is normally absent; this, too, in spite of observation of a large number of healthy individuals. I regret that my experience does not coincide with his. Among thirty adults I have encountered two in whom this phenomenon could not be evoked in any manner whatsoever, or in any place, either by a light tap or a stout blow, either above or below the patella, either at one time or at another. Among twenty-three children it has been my fortune to meet with one, in whose one leg it was not to be gotten, and in whose other leg it was very weak. Bannister* failed to find it twice among about thirty-six healthy individuals, mostly adults.

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* Op. cit.

In the great majority of adults I have ascertained that the tendon-reflex could only be produced by tapping about one inch below the lower edge of the patella, either at or slightly to one side of the median line; whilst in a small number the characteristic forward jerk would follow a blow upon the tendon at any point over a larger surface, this surface sometimes extending from the patella to two inches below. The most usual site in children — fifteen out of twenty-three — whence alone the tendon-reflex can be evoked, is on the lower edge of the patella; in a lesser number it is confined to a spot just below this, or it is slightly diffused. The variations in the strength of the manifestation were wide, being in some cases, as I have said, absolutely nil, in others weak, in still others violent; indeed, the normal limits touch, upon the one hand, the pathological absence of locomotor ataxia, while, upon the other, they attain to the pathological exaltation witnessed in the moderate cases of spasmodic spinal paralysis.

In somewhat over one-half of the subjects—eighteen out of thirty-two — the tendon-reflex was not of equal strength upon the two sides. The difference was slight in one-half the cases, strongly marked in the other moiety. There was apparently no constant preponderance of strength or weakness upon either side.

The fact seems to have been overlooked that the tendon-reflex of the quadriceps extensor, whatever be its value, can only be of use in a disease involving the lower extremities, and only indirectly, if at all, of service in affections of the upper limbs. The triceps is to the humerus and the ulna what the quadriceps extensor is to the femur and the tibia — the great extensor muscle of the limb; so forcibly, indeed, did the analogy impress the mind of Cruveilhier that he described the latter by the name of the triceps femoralis. It has occurred to me, therefore, that the triceps, being the analogue of the quadriceps, might be utilized for diagnostic purposes in the upper limb, as the other has been used in the lower. Like its analogue, it terminates in a large flat tendon, superposed upon a bony base, and thus favorably shaped and situated for the manifestation of the phenomenon of which I am treating. In eighteen subjects whom I have examined, I have found the tendon-reflex of this muscle always present. It is not, however, so easily evoked as in the quadriceps, a smart, quick blow with a percussion-hammer being necessary to its production. The strength of the manifestation has been moderate, varying but slightly, and far less than in its fellow of the leg. In examining this muscle, I have supported the arm of the subject at a right angle to the trunk by my hand placed in the hollow of his elbow, taking care that the forearm hangs passively; and I have always tapped upon the bare skin.

The physiology of the tendon-reflex has been a much-mooted problem. I have endeavored to determine for myself whether it is really a reflex action, or whether it is due to an inherent muscular irritability. For this purpose I have made a number of experiments upon rabbits. In these animals the tendon-reflex of the quadriceps extensor is easily obtained. I have always laid them upon the back; then flexing the leg upon the thigh, in which position the animal will generally permit it to remain, and grasping the thigh with the one hand, I have lightly tapped with the other by means of a percussion-hammer upon the quadriceps tendon as it is stretched across the anterior surface of the knee like a narrow drum-membrane; at the same time I have been particular to discriminate between the resulting quick, sharp, jerky jerk of the foot, and the heavy, inert movement of the leg and foot as a whole that may be produced in the delicate limbs of the rabbit simply by the shock of a blow, without any muscular action whatever. Ascertaining in each individual animal the presence of the tendon-reflex, I have broken up the spinal cord between the different lumbar vertebræ, and observed the effect. *I have satisfied myself that destruction of the cord between the fifth and sixth lumbar vertebræ instantly causes the tendon-reflex of the quadriceps extensor to cease.* Strike now as strongly as you will upon the tendon, there is absolutely none of that sudden movement of the foot which was so marked a few moments before. An animal upon whom this operation had been performed was kept alive for three days, but there was not witnessed any return of the phenomenon. Sections of the cord between the other vertebræ increased the tendon-reflex of the quadriceps, simultaneously with the other reflexes. These experiments are repetitions of those made by Tschirjew,* and confirmatory of them. Schultze and Fürbringer † had previously demonstrated that section of the crural nerve also put an end to the tendon-reflex of the quadriceps.

The tendon-reflex of the quadriceps extensor is therefore truly a reflex. In other words, for its manifestation it is necessary that there should be a structural integrity: *a.* of a certain portion of the spinal cord, probably of its grey matter, what is called in the fashionable phraseology of modern science, a *centre; b.* of certain nerve fibres conducting impressions inward to the cord, *afferent* nerves; *c.* of certain nerve fibres conducting actions outward from the cord, *efferent* nerves. What is true of one muscle is probably true of all. What is true of the muscles of rabbits is probably true of similar phenomena in human muscles. As the crural nerve in the rabbit takes its origin, according to Krause, in the spinal cord between the fifth and sixth lumbar vertebræ, as it is distributed to the quadriceps extensor, and as section of it and of this segment of the cord destroys the tendon-reflex, it follows that the afferent and efferent nerves of the tendon-reflex of this muscle run within the sheath of the crural nerve. Are they, then, the ordinary motor and sensory nerves, linked by the centre common to such nerves? After some experimentation and thought, I am prepared to meet this question with a decided negative. I am prepared to state my belief that the nervous apparatus of the tendon-reflex—its spinal centre, its afferent and its efferent nerves—are entirely distinct from the nervous mechanism of the ordinary motor and sensory nerves.

My reasons for this belief are as follows:

1.—*The nerves possess a different excitability from that of the ordinary motor and sensory nerves, and are animated and depressed by different stimuli.*—I have repeatedly administered ether to rabbits to such an extent as to have the limbs become perfectly flaccid, at the same time that the deep thrust of a knife-blade produced not the slightest movement—in brief, to such an extent as to have absolute motor and sensory paralysis—whilst the tendon-reflex of the quadriceps was undiminished, or even increased in strength. I have repeatedly obtained similar results with chloroform. Westphal and Erb have completely paralyzed the motor nerves of animals by means of woorara without injury to the tendon-reflex. All the German observers concur in stating that it cannot be evoked in any other manner than by tapping upon the tendon. Heat, cold, chemical irritants, electricity—unless a strong current be applied—fail to call it forth. I have observed one striking fact, germane to this particular, in all my experiments: When the spinal cord with its membranes is laid bare, there ensues a short period of extreme shock in the animal, during which there is complete loss of motion and sensation, even though no anæsthetic have been employed; it is, indeed, precisely at this period of painlessness that Claude Bernard advises the physiologist about to operate upon the nerve roots to isolate the posterior one, rather than wait until its sensibility is restored and the movements of the animal offer serious hindrance. But at this period the tendon-reflex, far from being abolished, is actually increased in strength! Tschirjew* exposed the crural nerve, encircled it with a light string, gently raised it upon the string, and thereby abolished the tendon-reflex of the quadriceps. There was, however, no symptom of any interference with ordinary motility or sensation, so far as could be inferred from the facts that the muscle was not appreciably relaxed and that the sensibility of the exposed nerve had not been altered. Its absence or its increase in the healthy, moreover, is not attended by any appreciable derangement of the ordinary nerves. Pathological facts, scanty as they are, are not at variance with the physiological. Whether the tendon-reflex be univerally absent or not in *locomotor ataxia*, it is certain that it is wanting in a certain proportion of cases, and at a stage long anterior to the onset of any paralytic phenomena. On the other hand, it is greatly increased in spasmodic spinal paralysis, a disease which is not, as a rule, accompanied by any hyperæsthesia, and in which there is not infrequently a slight anæsthesia. Muhr[*] affirms also that it is generally present in general paresis.

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* Op. cit.

† Op. cit.

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* Op. cit.

2. *These nerves are either not at all or imperfectly under the control of the will.*—Tap at the proper point upon the quadriceps tendon of a strong man. and he will be unable to prevent the immediate forward jerk of the foot. I have had an opportunity of testing this particular in an exceptionally favorable individual. There is a certain Mr. Mann, well known of late to the profession of New York and other cities, who possesses a remarkable and probably unprecedented control over the different muscles of his body and extremities. He can at will produce dislocations of the various joints by snapping the articulating surfaces of bones out of their sockets. He can so contract and expand his chest that the difference between the maximum expansion and the maximum contraction is thirteen inches. He can so contract his abdominal muscles as almost to lay the recti against the vertebral column. He can exactly simulate a talipes varus. But he cannot prevent the peculiar jerk of the foot, when I tap upon his quadriceps tendon. He can limit it by a simultaneous and violent action of the muscles of the calf of the leg; stop it, he cannot.

3. *It is probable that these nerves possess a much slighter sensibility than the ordinary sensory nerves.*—When the tendon-reflex is evoked in the human being by tapping upon the quadriceps, the blow which is adequate to produce what is oftentimes a violent muscular movement gives rise either to no sensation at all, or to a very faint one. Under no circumstances that have come under my observation, in the healthy or in the sick, is the blow felt as it would be if impinging upon an ordinarily sensitive nerve, as upon the ulnar at the elbow for instance.

4. *These nerves do not possess the same conductivity as do the ordinary motor and sensory nerves.*—Burckhardt[†] and Tschirjew[‡] are at some variance with each other in regard to the actual rate of transmission of the nervous current in these

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[*] Op. cit.

[†] Op. cit.

[‡] Op. cit.

fibres, but they both agree in stating that the length of time intervening between a blow upon the quadriceps tendon and the ensuing jerk of the foot is greater than would be ordinarily consumed by the passage of a nervous impulse inward along a sensory nerve to the spinal cord, through the spinal ganglia, and outward along a motor nerve to the quadriceps. The explanations offered by these writers are very unsatisfactory and contradictory. Burckhardt makes the singular and incomprehensible suggestion that the time employed is sufficient for the passage of the nervous impulse through the lumbar plexus or the spinal ganglia, or for the direct irritation of the muscle. Tschirjew inclines to the belief that the intraspinal paths of conduction of these nerves must be shorter than usual, or must be endowed with an unwontedly rapid conductivity. If my foregoing argument have any value, its statements should conduct us to the simple and satisfactory conclusion that these nerves possess a conductivity of their own, differing greatly from that of the ordinary motor and sensory nerves. In all my experiments I have been impressed with a fact that lends support to this view. A blow upon the quadriceps of one leg of a rabbit, be the tap ever so light, will not only evoke the tendon-reflex of the corresponding side, but will also bring forth in lesser degree that of the opposite knee, without causing any other muscular movement.

To sum up, then, it appears that these nerves differ distinctly from the voluntary motor and sensory nerve fibres in being affected in a different manner by the same influences, in being affected by other influences, in being but slightly under the control of the will, in being but slightly sensitive, in possessing a much more rapid conductivity. I have no doubt but that they are distinct nerves; and I would suggest for them the name of *tendon-nerve fibres, motor and sensory.*

It would seem evident from the preceding facts that the tendon-nerve fibres belong to the class of the involuntary nerves. This opinion is strengthened by further inquiry. Contrary to the experience of Erb and Westphal, I have succeeded in abolishing the tendon-reflex in rabbits by means of ether, chloroform and woorara. This I have done with ether and chloroform by pushing the anæsthetization until there were distinct symptoms of approaching paralysis of the involuntary nerve nuclei of the medulla oblongata, as evidenced in the most of my animals by the characteristic cry, entirely similar to the one which may be produced by touching a certain portion of the medulla with a stylet, or by a marked diminution and deliberation of respiration, as well as by extreme rapidity of the heart-beat. When this degree of narcosis is reached, and invariably when heralded by the peculiar involuntary cry, the tendon-reflex of the quadriceps has ceased. Removing the anæsthetic and allowing the animal to slowly revive, the tendon-reflex has remained absent for a period varying from a minute to a minute and a half, and has then suddenly returned in increased strength. At the period of this return, the respiration increases in frequency, to and beyond the normal, the while the cardiac pulsations are reduced in frequency; in a word, the return of function in the nerves of the tendon-reflex comes *pari passu* with the return of function in the pneumogastric. Chloroform produces this cycle of events more rapidly than does ether, and it also abolishes the tendon-reflex for a slightly longer time. When use is made of woorara, the tendon-reflex disappears at the moment of great diminution or cessation of respiration, and of tumultuous action of the heart; but whether it reappears or not I am unable to say, as my experiments with this drug have been fatal to the animals. It will be remembered that the tendon-reflex was exalted at the moment of opening the spinal canal and exposing the cord and its membranes to the air. Precisely at this moment, also, the respiration increases greatly in frequency and the cardiac pulsations decrease; that is to say, the functional activity of the par vagum is greatly increased. The increase of this peculiar reflex in spasmodic spinal paralysis is, furthermore, though not attended with any exaggeration of reflex action in the voluntary nerves, frequently synchronous with an increase of function in the involuntary nerves of the bladder, giving rise to the frequent micturition of this malady. These details, when considered together with the imperfect response to volition, the slight degree of sensitiveness, certainly seem to show a close relationship between the tendon-nerves and the class to which the pneumogastric belongs.

But, it may be reasonably asked, is there any proof that an involuntary nerve like the pneumogastric ever terminates in a voluntary muscle like the quadriceps? Certain experiments of Claude Bernard,* the peerless reporter of nature, are very significant in this regard; and it is difficult to understand how they could have failed to attract and maintain the interest of physiologists through so many years. The facial nerve, as is well known, in its passage through the aqueduct of Fallopius receives a branch from the pneumogastric. The great Frenchman cut this branch before its junction with the facial. Immediately the ear on the corresponding side became, as he describes it, "low and hanging," while the ear of the other side remained unaffected. The animal was irritated, and the hanging ear became erect. The animal was left in repose, and the ear drooped as before. At another time he removed the superior cervical ganglion of the sympathetic.† The orbicular muscle of the eye of the same side at once became contracted, and remained so. If the globe of the eye were touched, the orbicularis contracted still more, so long as the irritation was continued; but it could not be relaxed. In another dog a section was made of the sympathetic nerve belonging to the superior cervical ganglion, so as to obtain the same persistent contraction of the orbicular muscle. A drop of ammonia was now placed on the conjunctiva, the pain causing the animal to close the eye obstinately and constantly. The peripheral fillet of the cut sympathetic was stimulated, and, in spite of the pain, the eyes were opened widely. There would thus seem to be no question that within the sheath of the pneumogastric there run fibres which terminate in the muscles of the ear, as well as that within the sheath of the superior cervical sympathetic there run fibres which terminate in the orbicular muscles of the eye. It would seem to be equally clear that the branch from the pneumogastric to the aural muscles is motor. The doubt might naturally arise as to whether it did not cause the ear to stand erect by a vaso-motor action, by constricting or enlarging the vessels, by engorging the ear with blood or depleting it. But this cannot be, because,

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***Systeme Nerveux*, t. II., p. 40, Paris, 1858.

†Ibid, pp. 35, 472, 499.

in the first place, there are no appearances of anæmia or congestion about such an ear; in the second place, when the cervical sympathetic is cut, and the ear becomes plethoric, its attitude is unaltered; and, in the third place, when the cut sympathetic is irritated so as to remove the aural plethora, the aural attitude is still unaltered. As to whether the fibres from the cervical sympathetic to the orbicularis palpebrarum are motor, and not vaso-motor, is of itself perhaps not so certain. Nevertheless, the proof first adduced that a condition of extreme anæmia or extreme congestion in a muscle is not alone sufficient to produce any marked contraction or relaxation, going hand in hand with the proof that a purely motor nerve may run in a bundle of involuntary filaments, should warrant us in regarding it as very probable that the fibrils to the orbicularis are intrinsically motor.

These branches to the ear and the eyelid, however, have an action that is in a measure independent of the voluntary motor nerves of the same parts. As we have seen, when the auricular branch of the par vagum is severed, such irritation of the animal as will call forth a voluntary effort, which must pass through the facial, causes the ear to be erected. Section of the facial, on the other hand, has as a consequence a complete paralysis of the orbicularis palpebrarum, although the filament from the cervical sympathetic to this muscle is uninjured. Still again, this same section of the facial, although it be made before the junction within the aqueduct of Fallopius of the branch from the pneumogastric, leaves the ear pendant and flaccid. The involuntary and the voluntary motor nerves of the parts have each therefore, as it would seem, an action that is complementary the one to the other. Be it noted, that although the facial can erect the ear when the pneumogastric fillet has been severed, such erection remains only during the continuance of voluntary effort. Cut the facial, galvanize its peripheral end, and in spite of the cervical sympathetic not having been touched, the eyelids will be approximated. This would appear to mean that the voluntary fibre imparts to the ear and eyelid an attitude in which it is maintained by the involuntary fibre. May not the tendon-nerves, as they bear a striking air of kinship to the pneumogastric and the sympathetic, play some such part in the voluntary muscles of the other parts of the body? I cannot believe that it is irrational to assume that there may be nerves in every muscle necessary to keep that muscle fixed in a certain position without an effort of volition. The countless expressions of the human face, imparted to it by muscular action, unconsciously the same through years, asleep and awake; the different conditions of contraction or relaxation of the eyelids and nostrils in different individuals; the constant station of the ears in certain quadrupeds, the peculiar positions of the different limbs, the slight flexion and pronation of the forearm, the eversion of the thigh; would all reasonably demand a set of nerve fibres that should act automatically, without continuous impulse from the will, day and night, asleep and awake, as the heart beats, as the lungs respire, and still be subject to modifications by the action of the will. Should there be such fibres, their distribution to the tendon might, as we could conceive, and as Sachs* has claimed to be so in fact, answer best the purpose of their creation; because when the tendon is set in motion the whole muscle would contract. This particular point, however, I offer as a mere surmise.

In conclusion I desire to remark that the discussion during the last few years in Germany as to the existence of inhibitory nerves has brought to the light a few phenomena that may possibly have a bearing upon the views advanced in this paper. Nothnagel* made a section of a dog's spinal cord in the dorsal region. In the course of time there ensued strongly tremulous reflex movements in the hinder extremities whenever the animal was suspended in the air by the fore paws. The sciatic nerve was then exposed. Gentle pressure upon it instantly suspended the reflex movements; strong pressure upon it greatly increased them. Tschirjew, it will be recalled, laid bare the crural nerve in a rabbit, passed a string carefully around it, raised it gently, and thereby at once abolished the tendon-reflex in the quadriceps, without apparently affecting the motility or sensibility of the knee. Exactly of what import this coincidence may be, whether Nothnagel by his gentle pressure suspended the functional activity of the same set of fibres that Tschirjew stilled by his light lifting, I am not prepared to say. I would merely have the fact borne in mind. Yet, when I recall the quick suspension of the tendon-reflex by minute causes, I am disposed to believe that the fibres in the two cases were identical; and herein may be a possible clue to the comprehension of those finely tremulous, rhythmical movements which we daily witness in paralysis agitans and disseminated sclerosis, and which serve to distinguish them so strongly from the quickly jerky movements of wider range in chorea. It is among the possibilities, too, that the symptoms of locomotor ataxia and spasmodic spinal paralysis may be better elucidated than they are at present by a further study of these nerves.

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*Op. cit.

*"Beobacht. über Reflexhemmung." *Arch. f. Psych. u. Nerv.*, VI., S. 332. 1875.

ART. VI.—CASE OF INFANTILE ENCEPHALITIS, FOLLOWED BY ATHETOTIC SYMPTOMS.

BY EDWARD C. SPITZKA, M. D., NEW YORK.

TOWARDS the latter end of February last a young man presented himself to me for treatment, at the North-Eastern Dispensary, who exhibited a group of symptoms which, in their aggregate, I am disposed to class under Hammond's "Athetosis." It was first noticed, on examination, that there was considerable bodily asymmetry, the entire right side of the cranium and face, as well as the right arm and right leg, being noticeably smaller than the corresponding parts upon the opposite side. This asymmetry was most marked in the face and cranium; less so in the arms, and least of all in the legs; the muscles of the atrophic side were contracted and firm, and the right side of the face was drawn, its features being sharp, contrasting in this respect with the plump left side. The next peculiarity noticed was a constrained position of the right limbs: the arm was extended in a horizontal line behind the patient, and at the same time twisted outwards, while the leg was thrown forward, the foot inverted and adducted; the knee was flexed and somewhat in the position of a genu valgum; from the patient's expression of countenance — and this was confirmed by his subseqnent admission as correct — I was led to believe that he did not exert his will to maintain the limbs in their abnormal position.

On now seizing his arm, "untwisting" it, bringing it out horizontally, and letting it go, the member was agitated by violent spasms, which involved the muscles of the fingers, hand and forearm individually. These movements, after several pendulum-like oscillations, which gradually increased in intensity, culminated in the arm being violently struck against the hip, where, after several tentative efforts, it remained fixed. If the arm was drawn away from its support, it again became agitated, and, if it happened to strike behind the hip, it was violently drawn back in its original constrained position. During all this time the patient felt worried, and stated that he did not feel fully at ease unless his arm was thrown out behind him, in the manner above indicated.

In walking he habitually carries it in this position, attracting the notice of all passers-by; while he can walk many miles without fatigue, he has to halt every ten or twenty steps to "recover his feet;" that is, with each successive step there is a more pronounced inclination for his toes to become flexed under the foot, and this tendency extending finally to the foot, as a whole, forces him to halt until the flexing tendency subsides, which it does in from twenty to sixty seconds.

Thus far I have described only the coarser disturbances of motility and position; these were combined with other more delicate anomalies. Unless the arm was fixed in one of the two positions in which I stated it to be comparatively quiescent, the individual fingers, independently of each other, executed movements which I can compare to nothing so well as those described by Dr. Wm. A. Hammond and others under the name of athetosis. In the course of these movements the hand frequently assumed positions exactly like those represented in the wood-cuts in the former author's work.

An additional peculiarity noticed was, that while usually the hand was flexed in the wrist and the fingers extended as a whole, as soon as the latter were forcibly flexed, the hand became violently extended, and as soon as it had reached the limit of extension, the forearm underwent an equally involuntary supination and flexion. If now, on the other hand, I flexed the extended hand, the fingers once more became extended and separated ; or, if I extended instead the fingers, the hand became flexed. The transition was accompanied by athetotic movements.

When the patient's foot was liberated from the restraining influence of the shoe and stocking, his toes were seen to undergo athetoid movements also, but with much more regularity of rhythm and constancy than the fingers. There was also a similar relation between the relative position of the toes and of the foot, as a whole, as that just described in the case of the hand.

All the detailed symptoms formed a constant part of the patient's condition, and could be evoked at all times ; besides this there were paroxysmal symptoms of two kinds. The first kind consisted of spontaneous exacerbations of the clonic spasms, known as athetotic ; these involved the muscles of the face, fingers, toes, hand, foot, arm and leg of the right side, and ceased after having lasted an hour or even a whole afternoon. Mental excitement or motor over-exertion seemed to bring them on ; he had never had them at night, to his knowledge, unless frightened out of his sleep.

The second kind consisted of attacks of vertigo, during which the patient claims to be conscious of what is going on around him. He states that he has these attacks only in the summer season and when exposed to the sun. Subsequent developments showed that he has them in winter and independently of caloric influences as well. He also occasionally has photopsia of a remarkable character.

The lobules of both of his ears are congenitally defective ; none of his brothers or sisters have this deformity. There is stricture of the ductus ad nasum on the affected side, probably related to the asymmetry of the skull. He complains of paroxysmal pains in the left mammary region.

His mental condition is fair, considering the circumstances of a necessarily defective education, and although his memory is not very accurate, he seems to possess a considerable share of good sense, abstaining from alcoholic liquors because he has seen its bad influence upon members of his family. He has taught himself to write with his left hand, though clumsily.

As Doctor J. G. Kiernan, of this city, had been kind enough to refer this interesting case to me, and as the patient himself was unable to give me all the history I desired, I asked my correspondent to inquire as to the family and personal history of the patient, members of whose family were under the Doctor's charge. I received the following reply:

* * * * "The hereditary history is unfavorable on both sides, especially on the maternal side. The maternal great-grandfather, great-granduncle, grandfather and two uncles died in apoplectic attacks; on the paternal side the relatives have been long-lived, but the father himself was very intemperate for many years before, and at the time of the patient's birth. The patient is one of twelve children, three of whom were still-born, one died a day after birth, and five are still living. Of the latter, a sister is subject to vertigo, accompanied by a hissing sound in the right ear. Although I have not examined her myself, I should judge from the fact that these attacks have a connection with the menstrual periods, and that it has been found necessary to remove her from school on their account, that she is epileptic.

"The patient I sent you, M. H., is twenty-two years old, Scotch descent, and is stated to have been perfectly healthy and intelligent up to his fifth year, when he passed through an attack of scarlatina. While convalescing he was one day standing in conversation with his sister, when he suddenly fell down unconscious, after uttering a loud scream. His consciousness returned after about one hour, and projectile vomiting now came on, which lasted until noon of the following day. I say projectile, because it was graphically described by the mother and older sister in such a way as to leave no doubts in my mind.

"When the vomiting ceased, it was discovered that the patient was paralyzed on one side of his body (the right) and unable to walk. After several months he gradually began to regain power on the paralyzed side, and except for the peculiar (athetotic) movements which subsequently developed, he seems to have regained motion nearly entirely.

"I cannot find out how long ago the movements you speak of first commenced; it seems that for many years he was entirely free from them; that they developed slowly and gradually, and that they attained their present degree of development about three years ago. The attacks of vertigo I have myself witnessed this winter, and the patient presents a stupid appearance after the attack is over, so that I consider an epileptic nature not out of the range of possibilities. While his general mental condition is good, his relatives state that he is fond of relating imaginary occurrences. His memory is defective; he is a poor arithmetician, and has the tastes and disposition of a boy of fourteen. I do not believe that there is the slightest ground for suspecting any specific disease in the family."

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From this I have seen reason to designate the case as one of infantile encephalitis (Griesinger), followed by contractures and athetosis. When exhibiting the patient before the Neurological Society I asked Dr. E. C. Seguin, the only one present at the meeting who had seen Hammond's original case,* how far M. H. resembled the latter. He stated that in several respects the movements seemed to be different, and further, that he had several times himself observed symptoms following hemiplegia in children which, because they were similar to, but not identical with those manifested in Dr. Hammond's patients, he designated as athetoid. Since the term athetosis is only employed to designate a complex of symptoms, and since, moreover, the total number of cases presenting it is a small one, I should be loth to choose between two terms here, especially as a perusal of various cases scattered in the literature of the subject shows that there are many variations from the typical picture as presented by the first cases described.

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*Shown at the first session of the American Neurological Association, and not seen by the writer.

In addition to the slow clonic spasms, my patient exhibited the detailed interesting anomaly in the position of his limbs. Although not exactly like the peculiar constrained posture of epileptic imbeciles described by Meynert, and of a case of multiple syphiloma of the brain referred to by myself in this journal, yet it seems to be analogous as far as it goes, and I should attribute it to a similar lesion, namely, a sclerotic cicatrix or cicatrices in and about the thalamus and crus, regions whose injury in animals will produce corresponding anomalies of limb position. It is known that the athetotic symptoms themselves are more frequently found associated with lesion of the thalamus than of any other part of the encephalon (Gowers, Charcot, and others), and we may well suppose that in the present instance one extensive lesion, the residue of the post-scarlatinal encephalitis involving this region, is the cause both of the contractures and of the athetosis.

The patient's heart is very irritable, but neither colleagues who examined that organ nor myself could detect any unnatural sound.

Although he has been under my care for only a month, yet I think there is some improvement in his condition, especially in the coarser movements, which are less excursive and spasmodic than they have been. The athetoid movements themselves seem more intractable. My treatment consists in advising the patient to exercise the arm first with weight, and then to exercise his will-power *gradatim* on the affected muscles. In this latter respect he has made some progress. Ergot and conium are given on other indications to the same patient.

Nothing can be clearer than the difference between the clonic spasms exhibited by this patient and the post-hemiplegic chorea, with which some are attempting to confound athetosis. While convinced with Dr. E. C. Seguin that my case is not identical with those described by the discoverer of athetosis, I have ranged it under this head because otherwise it would be necessary to erect an entirely new symptom group for the reception of this single case.

I have seen two other and more typical cases of athetosis in patients suffering from paralytic insanity. It was discovered in them about three years ago by Dr. J. G. Kiernan, then of the City Asylum, and although the discovery was published without specified acknowledgment, and, in general language, by an asylum physician who made use of Dr. Kiernan's notes in the compilation of his paper, I think that the priority of discovery of this hitherto unknown complication of paresis belongs to Dr. Kiernan, who will shortly publish the details of his two cases in full.

ART. VII.—RESEARCHES ON THE SPINAL CORD.

BY ISAAC OTT, M. D.

THE spinal cord, besides being a conductor to and fro of nervous impulses, is also the seat of several nerve centres, administering to the functions mainly automatic. I shall consider in this paper only the practical bearing of certain points connected with the physiology of the spinal cord. These points have been worked out in the laboratory at Johns Hopkins University, and will be published in detail elsewhere.

SECRETORY FUNCTIONS.

That the spinal cord and medulla oblongata preside over the secretion of sweat has been shown by the researches of Luchsinger. These centres send out fibres in the cat through the anterior root of the fourth dorsal nerve, into the stellate ganglion, and then into the ulnar and median nerves to the skin of the anterior extremities. The sweat fibres for the posterior extremities run through the anterior root of the three last dorsal nerves into the abdominal sympathetic, and then into the sciatic. The anterior roots of the last lumbar and first sacral also send fibres into the sciatics. On the sweat glands are seated ganglia inhibiting the activity of the sweat secretion, and these ganglia are called into exaggerated activity by irritation of the fibres contained in the abdominal sympathetic. These inhibitory ganglia may serve to explain the phenomenon of a "hot non-sweating skin." Heat, ordinarily, is one of the greatest excitants of the sweat secretion, and ought to start the secretion in feverish states, unless there is some force inhibiting it. The most important question is, where is the seat of the sweat centres and the course of the sweat fibres in man? To decide this, pathology must be the field in which to find facts to elucidate these points.

SEAT OF SWEAT CENTRES.

Traube relates the following case, which has an important bearing on this point. Case I, R., aet. 51. Previously not sick, with the exception of an intermittent; a large, well-built frame, hearing good, no otorrhœa, no headache, both arms completely paralyzed and extended on the trunk, but could be passively bent at the elbows; the fingers, with the exception of the thumb, were bent into the hollow of the hand; there was complete anæsthesia, except about the shoulders; the lower extremities were like the arms, voluntarily immovable, bent at the knees, the tendons being on the stretch; reflex movements were able to be excited; the skin of both lower extremities without sensibility; skin of abdomen insensible; incomplete sensibility in the lower part of the thorax; sensibility of upper part of breast, throat and face undisturbed; no alteration in the sense of taste; no palsy of the face; urine passed involuntarily, and constipation existing.

In the last eight days before death he suffered from a great increase of sweat over the whole body. On autopsy the brain and medulla oblongata were intact, but about half an inch below the medulla the spinal cord was very soft, and three and a half inches below the pons the consistence was extremely small, the nerve substance being completely broken up. This part was strongly compressed by a tumor the size of a pigeon's egg, and attached to the anterior wall of the spinal dura mater by the anterior surface, whilst the posterior convex surface projected into the cavity of the spinal canal and against the anterior columns, without being connected with them. In the vicinity of the seventh cervical vertebra there was a very considerable softening, which was of greatest intensity upwards for about two inches. On a transverse section of the upper part of the cervical portion of the spinal cord, the surface was somewhat pale. In the softer parts the substance issued forth in a profuse manner, most so in the vicinity of the fourth cervical vertebra, but even here the pale red grey substance could be well distinguished from the jelly-white substance. The remainder of the cord was normal.

This case proves that in man a nutritive lesion in the cervical part of the cord can call the sweat centres below it into exaggerated activity. In a case of progressive muscular atrophy under my care, where the disease had extended to the medulla oblongata and was accompanied with profuse salivation, I noticed on the face an augmented amount of sweat, which indicated that the sweat centres extended up into the medulla oblongata. Further, there are several cases on record where a myelitis has abolished sweating below its seat. Thus, Mr. C., who had a localized myelitis in the dorsal region, with complete loss of sensation and voluntary motion, no sweating was observed in the lower extremities till voluntary motion and sensation returned. As to the path of the sweat fibres in man, pathological changes afford brilliant evidence. Thus, Dr. Pepper reports the following cases:

Case II. A patient was under observation for eighteen months, suffering with an abdominal tumor of either an aneurismal or cancerous nature. One of the symptoms most constantly complained of was profuse sweating, so that although he was a cleanly person an unpleasant exhalation could not be prevented. It is a remarkable fact that, by the patient altering his position, so as to alter the pressure, the sweating could be confined to one side.

Case III. Dr. A. H. Smith, patient, reported by Dr. Pepper. The person had an encephaloid cancer of the abdominal lymphatic glands, at a point behind the pancreas and corresponding to the seat of the solar plexus of the sympathetic. The diseased glands formed a large flattened mass, closely adherent to the vertebral column posteriorly, and to the pancreas anteriorly, although it was impossible, owing to the circumstances of the autopsy, to make a careful dissection of the nervous ganglia involved in or pressed upon by the cancerous mass. Here profuse sweating existed.

These cases give ample proof that in man the sweat fibres run in the abdominal sympathetic, and that, reflexly, they can call out perspiration in other parts of the body.

When we examine the course of the sweat fibres in the thorax, we find that some run in the sympathetic. Thus, Case IV.,* a cachectic individual, had either an aneurismal or malignant tumor at the base of the neck, which had grown up from within the thorax. The tumor was on the right side, and accompanied with profuse unilateral sweating on the right side of head, terminating abruptly at the median line. The right pupil was also contracted, and no thermometric observations were made. Here the pressure on the sympathetic has paralyzed it, and the pupil contracts just as when the nerve is divided. This case beautifully demonstrates the fact of sweat fibres running in the thoracic and cervical sympathetic.

If we now look up the cases of sweating in the arm, it will be found that sweat fibres undoubtedly run in the nerves of that extremity. Thus, Case V.† A man æt. 38, had for five years a body the size of a pea, very painful, seated in the substance of the deltoid muscle, which was excised. After a year the pain returned in the same place, and in the succeeding three years the pains were unsupportable. These pains were continuous, but had irregular exacerbations lasting from five to ten minutes, when the pains sometimes ran toward the forearm to the fingers, and at other times in the vicinity of the supra-spinous fossa. The left arm is able to be used for strong effort, but is much weaker than the right. About two inches below the head of the humerus there is found a scar 1½ lines in extent, at whose lower end is a knot, extremely sensitive, and the size of a bean. By touching it a benumbing feeling is felt in the hollow of the hand where, as well as in the axilla, a large number of drops of sweat are to be seen, whilst the same neighborhood on the right side of the body is dry. Schuh considered it to be a tumor of connective tissue on the circumflex nerve. He excised the swelling with a small segment of the thickened nerve. The patient recovered but the sweating persisted.

In another case (VI.),‡ J. M., æt. 26. In May, 1866, the patient noticed a small tumor on the left humerus in the neighborhood of the ulnar side of elbow-joint, which was movable under the skin, and opened, since which time the skin was always moist and covered with a scab. Here the sweating was chiefly in the volar side of the forearm and the volar side of fourth and fifth fingers, and ulnar side of the third finger, whilst the remaining part of the arm was dry. Here the tumor involved the ulnar nerve and kept up a continuous irritation of the sweat fibres contained in it. These two cases leave no doubt that in the ulnar and circumflex nerves are found sweat fibres.

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* Bartholow.

† Nitzelnadel.

‡ Nitzelnadel.

As to inhibiting fibres, Schuh has noticed in two cases that after section of the frontal nerve of one side sweating was continuous in the parts supplied by the nerve.

REFLEX SWEATING.

I now shall give cases where sweating is a reflex act.

Case VII.—Dr. Pokroffsky reports: A patient, male, presented the following appearances whenever he began to eat: The right side of the face, the greater part of the forehead, a part of the region about the shoulder, and the entire neighborhood of the temple, the ear, a little strip behind the ear, and the upper part of the neck on the right side became decidedly red and covered with a profuse perspiration. This collected gradually in large drops and, if not wiped away, would run down over the body. The right half of the breast, the right arm and leg were, during eating, somewhat warmer and more moist than the same parts on the left side. During the perspiration the right temporal artery became prominent and pulsated markedly. Pulsation was also noticeable on the inner side of the cheek, the pupils contracted equally, there was slight asymmetry of the face and tongue, the right eyelid could not be raised quite as high as the left. During eating the temperature rose 0.2 to 0.4 C. in the right arm-pit, and other variations showed themselves later. Irritation of the sympathetic by the induced current during the patient's quiescence produced no effect similar to that brought on by eating, but if the patient began to eat while the stream was passing, the usual symptoms were observed. Faradization of the cervical sympathetic during eating caused rapid diminution of the redness, and cooling of the right side. Various investigations were made, and showed the nervous system to be apparently normal. Electro-sensibility of the right cheek was diminished. The imperfect hearing was worse on the right side. There was enlargement of the liver and spleen, and the sweating dated back to a fever which was accompanied by parotitis of the right side.

Case VIII.—Mrs. H's child, when nursing at the breast, sweats at the knees—verified by the mother, an intelligent lady.

There are several cases on record where sweating ensues on one side of the face during eating. There seems to be in several of them a connection between four states: pre-existent parotitis, eating, sweating, and dilatation of the blood-vessels on one side of the face. What connection the parotitis may have I am unable to say, unless it has made the facial nerve more excitable, for this nerve when irritated calls out sweating in the face. The reflex circle here is the nerve of taste carrying sensory impressions to the medulla, which calls into exaggerated activity the sweat centres and the vaso-motor centres. In cats with cerebrum destroyed, electric irritation of the tongue brought out sweat in the anterior extremity.

ACTION OF MEDICINES ON SWEAT GLANDS.

Mr. G. B. Wood Field* and I have proved that muscarin calls the sweat secretion into activity by a peripheral action on the nervo-glandular apparatus. Since I have ascertained that when the sciatic has been divided for a week, and has completely lost all power upon electric irritation to call out sweat, the administration of muscarin will set the sweat glands into activity. Jaborandi under similar conditions has no power like this, although acting peripherally when the nerve is freshly divided. The inference is that muscarin and jaborandi act on different parts of the nervo-glandular apparatus. Atropin also retards the action of muscarin on the sweat glands.

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* *Journal of Physiology*, 1878.

RHYTHMIC FUNCTIONS.

When the spinal cord in the cat is divided completely at any height, the sphincter ani takes on a rhythmic movement similar to the diastole and systole of the heart. I have discovered that the sphincter vaginæ in the same animal and in the dog takes on a similar rhythmic movement when sections are made through the base of the brain from before backward; it is found that a section just behind the optic thalami is followed by a rhythmic movement of the sphincters. If, now, an irritation is made by the forceps of either the rectal or vaginal mucous membrane, one or both of the posterior extremities will set up a series of rhythmic movements, provided the spinal cord has been divided in the dorsal region. If, now, the spinal cord can set up rhythmic movements of the muscles, I cannot see why the rhythmic movements seen in man, of one of the extremities, are not due to either a central or reflex cause, acting on the central nervous system. Thus, the following case, reported by Dr. S. W. Mitchell, seems to me one where the nervous system has set up a rhythmic movement: "R. C., æt. 33; watch-case-maker; married, and has healthy children; no syphilis; his family history is bad — a sister, two aunts and his mother have had palsies in middle life; an uncle had epilepsy, and a cousin dementia. He was always nervous and excitable; used no spirits, smoked moderately, and was always well until the summer of 1865, when he was two days unconscious from sun-stroke, and has ever since been made weak by the heat of summer. About two years ago, in the summer of 1874, he observed that, while walking, the legs felt weak, and he was obliged to stop, as if to regain power, after which the right leg would drag for a time; at the same time he began to have pain in the back of the neck and lumbar spine. These pains continue. In Jan., 1875, he noticed a slight but increasing tremor in the left arm, and in two to three months the strange condition, which I shall try to describe. * * * * * * In sleep there was no movement; when he awakened he was conscious of the left hand being rigidly closed. In a few moments it began to twitch, the fingers moving as do those of a violin player. The slightest movement of any other limb, speaking or eating, even if he be fed, causes the left arm to execute a constant motion of striking the bed or his side, the limb being the while extended. When he arose and walked this action became more violent, and so much resembled the steady, rapid movements of a pendulum that I spoke of it at once to my assistants as a case of what might be called, descriptively, pendulum spasm. In fact, its rhythmic regularity was astonishing. Dr. Sinkler timed it on one occasion as 157; and on several others I found it always exactly 160. It was as accurate as the heart in its motions, but certain things always increased either the power or the number of the motions. * * * * * He has the power to stop the spasms by certain manœuvres. If he seizes the left hand with the right, and flexing the left arm, holds it, there is a kind of general spasm; the left hand, for a moment, seems to struggle with increasing violence; he totters, the face is convulsed; there is horrible pain in the back of the head. Then he gently releases the left arm, which, save for a slight tremor or twitching of the unquiet fingers, remains at rest, and may not move in violent spasm for an hour or more, and is sometimes nearly still for twelve hours." It has been found that the rhythmical movements of the sphincters can be arrested by an irritation applied to the sciatic. It would seem that, in the just described case, a sensory irritation (as grasping the arm) could arrest the rhythmical pendulum spasms.

VASO-DILATOR CENTRES.

That vaso-dilator nerves exist has been proved by the researches of many observers. Spinal vaso-motor centres also are quite generally admitted to exist. I have discovered that the skin of the pulps of the posterior extremities in the cat is innervated by vaso-dilator centres seated between the tenth dorsal and the first lumbar vertebræ. It seems to me that this fact will explain part of the symptoms in the cases in erythromelalgia described first by Dr. S. W. Mitchell. These cases must partly have a lesion seated in the spinal vaso-dilator centres. Dr. Mitchell describes the vasal symptoms as follows: "The next striking peculiarity of this disorder is the flushing of the part upon exertion. This symptom, which is usually absent in the very early stages, is a notable feature of the worst of the prolonged cases, and, in some mild instances, can always be brought on by great exertion of the foot. In the graver examples the area of greatest pain in the soles or hands is distinctly and permanently marked by a dull, dusky, mottled redness, as if the smaller vessels were always over-distended."

PATHS OF CONDUCTIVITY.

In the lateral columns of the dorsal region of the spinal cord of the cat run at least five kinds of fibres: sensory, motor, vaso-motor, sweat fibres and inhibitory. The vaso-motor path was determined by making a section of the lateral column and observing the color-changes in the posterior extremity on that side, and comparing them with the opposite side. Of course the feet were unpigmented. The inhibitory fibres were traced by making with Woroschiloff's instrument sections of the cord, to different extents, and observing if the rhythmic activity of the sphincters took place. It was proved that the inhibitory fibres arose in the optic thalamus and passed down the lateral columns of the cord to inhibit the rhythm of the sphincters. Flechsig has shown that it is probable that fibres arise in the optic thalamus and traverse the lateral columns, but has not stated their nature. If, now, fibres running in the lateral columns inhibit the ano-spinal centre, it is highly probable that fibres inhibiting other reflex actions also exist. The exaggerated reflex seen in cases of spastic spinal paralysis may be explained in this manner: The sclerosis of the lateral columns, removing the inhibitory power coming from the brain, allows the spinal reflexes to come into activity.

VAGINO-SPINAL, ANO-SPINAL AND VESICO-SPINAL CENTRES.

The ano- and vagino-spinal centres in the spinal cord are located below the vesico-spinal. The existence of a vagino-spinal centre explains those cases of vaginismus where the peripheral irritation causes a spasm of the sphincter. The treatment of those cases is, to remove the peripheral lesion, and depress the reflex activity of the spinal cord. The vagino-spinal reflex also preserves, or rather supports, the uterus, by keeping the sphincter in a more or less continuous activity, thus antagonizing the downward tendency of the uterus.

Reviews and Bibliographical Notices.

I.—RECENT WORKS ON INEBRIETY AND ALCOHOL.

I. HABITUAL DRUNKENNESS AND INSANE DRUNKARDS. By John Charles Bucknill, M. D., Lond., F. R. S., Fellow of the Royal College of Physicians, Late Lord Chancellor's Visitor of Lunatic Asylums. London: Macmillan & Co., 1878.

II. ALCOHOL: ITS USE AND ABUSE. By W. S. Greenfield, M. D. New York: D. Appleton & Company, 551 Broadway, 1879.

III. THE NATURE AND TREATMENT OF INEBRIETY. Also, the Opium Habit and its Treatment. By Dr. Edward C. Mann, Superintendent of Sunnyside Medical Retreat, Washington Heights, New York. New York: Charles A. Coffin, Steam Book and Job Printer, 87 John Street, 1878.

The three small volumes whose titles are given above, though all relating more or less to the uses and effects of alcohol, as found in fermented and distilled drinks, are nevertheless widely different in their objects, style and sentiments. The first is a duodecimo volume of one hundred and three pages, on good paper and fair type, and filled with a series of addresses and articles written by the author at various times from 1875 to 1877, and originally published, some in the *Journal of Mental Science*, some in the *Contemporary Review*, and others in the *London Times*. The eminent and learned author visited this country in 1875, and took special pains to acquaint himself with the condition and practical working of our Asylums and Hospitals for the Insane, and also to some extent, with the practical results obtained in such institutions as had been established for the treatment and reform of habitual drunkards, generally called Inebriate Asylums. He attended the annual meeting of the Association of Medical Superintendents of Asylums in America, for 1875, and his brief speech before that Association constitutes the introductory article in this volume. After his return home, the following year, he stated briefly the results of his observations concerning the latter class of institutions, in a speech made at a meeting of the Rugby Temperance Association, which appears to have led to all the subsequent correspondence and articles, and gave to them somewhat of a repetitional and controversial character. But neither the sharp controversial style of the author, nor the few verbal errors committed, such as substituting the name of

Dr. Walker for that of Dr. Turner, as the founder of the Asylum at Binghamton, and the spelling of the last name with a "p," have any general public interest, though they detract somewhat from the pleasure of the reader. The three leading and important topics discussed by Dr. Bucknill in this little volume, are: First, the question whether DRUNKENNESS is a disease or a vice. Second, the proper treatment of habitual drunkards, involving a consideration of the value, or want of value, of American Asylums for the cure of inebriates. And third, the relations of intemperance to insanity.

He acknowledges the difficulty of always maintaining clearly the distinction between vice and disease. On this point he says: "What is that distinction? Where is the *crux?* From the spiritualistic point of view the answer is easy; but what is the answer from our point of view—physiological?—As a guess at the truth, I would say that vice is a habit of the nervous centres of energizing in an emotional direction, mischievous to the well-being of the individual and of the community, but consistent with healthy nutrition, and not necessarily tending to diminish or destroy the vital activities of the individual. Disease I would define as a condition of some one or more parts of the organism, inherited or acquired, which always involves and implies an abnormal state of the nutrition of those parts, and does necessarily tend, if prolonged and increased, to diminish or destroy the vital activities of the organism." And, he adds, the first is to be managed by "education and reformation," and the second by "preventive and remedial medicine."

After eliminating two groups of rare cases, in which the action of alcoholic drinks coöperate with certain morbid conditions of the brain, to produce actual mental derangement or insanity, Dr. Bucknill comes directly to the question whether simple habitual drunkenness is a *disease* or a *vice*, and proceeds as follows:

"To many people the answer will be very simple, seeing that they will consider disease as a condition of the material, and vice as an affection of the immaterial element of our nature; but such is not my view, and I shall endeavor to consider both terms of the question by the same physiological method. And here it is needful to premise that as there is no such thing as a disease in itself, so there is no such thing as vice in itself. Doctors have fallen into the habit of realizing and classifying diseases as if they were all composed of various *materies morbi*, and were actual things; nay, sometimes they almost personify them, as in the old trope of "the pestilence which walketh in darkness," and moralists have done quite as much for vice. But the idea of a disease, be it ever so specific, can only be a mental creation generalizing diseased bodies, and of a vice the same, generalizing vicious beings. Reality ever attaches to the individual; and here let it attach, say to John Jones, and the question be whether said John Jones being often drunk, is diseased or vicious? I think it must be admitted, that when said John Jones is actually drunk his organism is in a state of disease, *pro tanto*, as the same must be admitted of him when he has eaten more than he can easily digest, or when he is exhausted by debauchery, or actually suffering disturbance of healthy function from any other sensual excess; for any condition of the organism by which its healthy functions are disturbed, and which, if indefinitely prolonged and increased, would tend to their suspension and even to death, must be called a diseased condition. The contracted question then comes to be, whether John Jones is diseased or vicious while he is still sober, and when he is about to drink the 'brewed enchantment,' knowing all the effects which it will have upon him?" After stating some of the causes and qualities common to both disease and vice, he proceeds to differentiate them as follows:

"*a.* Disease consists solely and entirely in some change in the organization, which is often known to, and is always thinkable by, the physician. This is a matter of certainty. But it is not known that vice consists in, or is even accompanied by, any such change. Certain vices may produce such a change, as an effect, but such changes are not known to exist as constituent conditions of vice; and if they do exist, they are too obscure for our present means of observation, or even for our power of thinking of them by analogy or imagination. If it be admitted that both vice and disease are dependent upon states of the organism, a consideration of one fact will suffice to prove that these states are very different. In the early stages of disease, and especially of nervous and mental disease, there may be no changes after death, appreciable by our senses or means of examination; but in late periods of such disease, such changes are mostly to be found. But in uncomplicated vice, whatever its duration and whenever death may occur, no such causal changes can be observed.

"*b.* The causes of disease are mostly physical, and the last link in the chain of causation—that is to say, the causal condition—is invariably so. Emotion may be the exciting cause, but there is always a physical link intervening, generally in the form of disordered innervation. The cause of vice, on the other hand, is always moral, even when the conditions of the vice are grossly material and sensual.

"*c.* The remedies for disease are mostly physical, and are invariably of a physical nature. Even medicine for the mind diseased, in what is called the moral treatment of insanity, is really directed to the material condition of the organ. The remedies for vice are of a different nature, and are mainly directed to elicit opposing desire, to make indulgence more immediately painful, and to influence the judgment.

"*d.* The successful result of remedies, which in one case we call cure, and in the other reform, is very different. The cure of disease establishes a healthy condition of the body; the reform of vice establishes a virtuous condition of mind, and even should the latter be looked upon as a condition of the emotional functions of the brain, the notion of it is different from that of merely physical health.

"*e.* With some exceptions, persons suffering from mental disease are not conscious of their misfortune. * * * * The vicious man is generally conscious of his vice, which, unless thoroughly brutalized, he regards with shame and remorse. Disease may occasion regret, but not remorse.

"*f.* Few men are diseased, but all are vicious. The one state is an accident to man's nature, the other is an element of it. And with regard to the vice of drunkenness, there was never yet found a tribe of savages so unsophisticated that they needed more than the opportunity, and the first lesson, to plunge headlong into the abyss. The first debauch may be the fault of ignorance, as perhaps that of Noah was, when he drank of the wine and was drunken; but the savages of all climes are habitual drunkards *en posse.* And the wild theory that habitual drunkenness is a disease, supposes that the existence of a keg of firewater will convert a camp of Indians into madmen, while even they have not yet tasted it, for while they are sober they will barter their possessions, their liberty, almost life itself, for its enjoyment. If we examine the condition of a man who has what is called an irresistible and uncontrollable desire to imbibe strong drink by these tests, we cannot fail to see that it is not that of disease, but that of vice. The passion for drink, however habitual and forcible it may be, is not associated with any physical change in the organism. The indulgence of it produces changes which generally lead the drinker through a life of misery to an early grave, but even this effect is not constant, and before it has taken place the organism of the drunkard may be, and sometimes is a thoroughly healthy one."

If this lengthy quotation is scanned closely, the reader will see that Dr. Bucknill makes a clear and very important distinction between the habit of drinking—the mental act determining the individual to drink—which he calls drunkenness, and the effects of the drink on the physical system of the drinker. The first he insists is in all ordinary cases simply a vice, or vicious habit. The second is a morbid effect, which may be either temporary or permanent, according to the frequency or persistence of the drinking. Hence, while he denies that ordinary drunkenness is *a disease,* he freely admits that many drunkards are diseased. As the real nature of drunkenness or inebriety is attracting much attention and is a subject of real importance, we cannot better express our views concerning it than by repeating what we said to the meeting of the American Association of Superintendents of Inebriate Asylums in this city (Chicago) in September, 1877. We then said, that the voluntary act of taking intoxicants sufficient to induce inebriety or intoxication, is a vice for which the individual is morally responsible, probably none will deny. That the condition of the brain, nerves and blood, which constitutes inebriety or intoxication, is a morbid or diseased condition of those parts is equally plain. Alcohol and opium are physical agents. When taken into the human system they are capable of so modifying the condition of the brain and nerves as to pervert their functions, constituting plainly a diseased condition. The habit of using intoxicants voluntarily and with a knowledge of their effects is certainly a vice which is to be corrected by mental and moral influences. The effects of such intoxicants on the human system just as certainly constitutes a diseased condition of the various organs and structures, which is to be removed by treatment on the same principles as we treat other manifest diseases of our physical organization. If we limit the word INEBRIETY to a designation of the latter, we leave no room for controversy in regard to the question of disease. If we allow it to include the former, we embrace under one name things essentially different, and at once open the way for misunderstandings and controversy. Another fruitful source of error consists in the confounding of a predisposition to a disease with the disease itself; as when we say such and such cases of inebriety are hereditary; that such a man was born an inebriate, &c.

This again startles the moralist, with the idea that if a man is born a drunkard, he cannot be held responsible for his vice, and the same old controversy returns upon us. It has seemed to me, we would avoid all such misunderstandings and make much better progress in the enlightenment of the public as to the nature, consequences and responsibilities involved in the cause of intemperance, if we were more careful to use only such words and phrases as convey our ideas accurately. I presume no one in speaking or writing of hereditary inebriety means actual inebriety at birth or during the period of early childhood. But simply that the child was born with certain weaknesses or susceptibilities which made it more easily contract the habit of using intoxicants, and more readily succumb to their influence during the progress of life. Hence, instead of saying he was "born an inebriate," or that the case was one of "hereditary inebriety," we would express the truth much more accurately by saying that he was born with a PREDISPOSITION to inebriety; or the case was one influenced by HEREDITARY PREDISPOSITION. My own observations through a period of forty years, on a field not very limited, have induced me to believe that even the hereditary predisposition to inebriety has been greatly over-estimated. The statistics on the subject have all rested on only a partial observation of the facts involved.

Whenever an inebriate was found to have had intemperate parents or grand-parents, he has been placed in the hereditary list. And when it was found that a larger proportion of the children of the intemperate became addicted to the same habit, than of the children of the temperate, it was regarded as conclusive proof that the disposition to use intoxicants was transmitted hereditarily. It requires but little observation, however, to show a very important source of error in these conclusions. The fact that children of intemperate parents are generally in direct contact with, and are taught to use these drinks even from their infancy, having thereby an opportunity to acquire a taste or love for them long before they arrive at maturity, is overlooked. And yet if we note the difference between the circumstances favoring the formation of an acquired habit and taste for intoxicants among the children of intemperate parents, compared with those surrounding the children of the temperate, we will find ample reasons for the difference in the results without even a recognition of hereditary predisposition.

Another mischievous fallacy is taught by some observers and eagerly adopted by many magazine writers, in the assertion that a taste or longing for some kind of stimulating or intoxicating agent is an inherent or instinctive quality of our nature; and consequently its gratification is a legitimate compliance with the designs of the Creator. The only proof ever adduced for such doctrine is the assertion that all nations and tribes have sought out and adopted the use of some article of this class. Now, if we analyze the facts involved in a careful investigation of this subject, we will find it readily resolved into this, that man was created with a susceptibility to feelings of anxiety, pain, sorrow, weariness, weakness, heat, cold, &c., and an intelligent desire to be relieved from all such unpleasant feelings.

This desire for relief prompts the intellect to seek means for affording such relief.

Neither the feelings themselves nor the desire for relief, suggest any particular remedy or class of remedies. They inherently or intuitively suggest no more idea of a glass of whiskey than of a glass of milk; no more thought of a mug of beer than of a basket of beans. But if by accident or design they happen to use anything that affords relief, then they will be likely not only to use it again themselves, but will recommend it to others. Thus the article used, whether alcohol, milk, tea, water, opium, cannabis Indica, is altogether a matter of experience or information.

Without pursuing these thoughts further, I may sum up the whole in the following brief propositions:

First, That the use of alcohol and all other anæsthetic and intoxicating agents, is founded primarily on simple experience, and not on any innate or natural instinct belonging to the human race; the only instinct involved in the matter being a desire for relief from unpleasant impressions, or the enjoyment of pleasant ones.

Second, The voluntary use of alcohol or other intoxicants is primarily a mere act of the will under the influence of motives, for which the individual is morally responsible just in proportion to his knowledge of the consequences of his act, and the character of the motives governing him.

Third, As alcohol and other intoxicants are physical agents, incapable of undergoing digestion and assimilation when taken into the living system, they alter the properties and functions of some one or all of the physical structures and organs of the body from their natural condition. In other words, they produce an unnatural, morbid or diseased condition. At first such morbid conditions are temporary, but by frequent and long-continued repetitions they may become permanent, constituting important and extensive changes both of function and structure, which no mere exercise of the WILL can control.

Fourth, When the physical changes indicated in the preceding paragraph have become well established in the inebriate, both analogy and observed facts point to the conclusion that children born of such parents will be more liable to have weak or defective physical and mental organizations, predisposing them to yield more readily to all morbid influences. If such children in the progress of life become in any degree exposed to the influences of alcohol, they yield more readily to its evil effects, as they do to the evil effects of any other injurious agents. In this sense, and this only, do we regard alcohol as capable of perpetuating a hereditary influence towards inebriety.

We fully agree with Dr. Bucknill, that the habit of drinking, or common drunkenness, is a vice and not a physical disease; but we do not think he fully appreciates the importance of the morbid changes in the brain and nervous structures produced by habitual and persistent drinking, and the effect they have in impairing the mental operations, and, consequently, the individual's power of self-control. Yet this has an important bearing on the next topic discussed by the author, namely, the proper treatment of habitual drunkards.

With a few rare exceptions of acknowledged insane drunkards, whom Dr. Bucknill thinks could be as well accommodated in regular asylums for the insane, he decidedly opposes all laws for the forcible restraint of the drunkard, or for the establishment of separate asylums for the reception and treatment of inebriates with a view to their *cure* as persons simply afflicted with disease. He severely criticizes and denounces as worthless failures all those inebriate asylums in this country, that are managed on the idea that inebriety or common drunkenness is a disease to be cured instead of a vice to be reformed. He says, "he visited many of the American inebriate asylums, and he came to the conclusion that the gentlemen confined in them were generally rather proud of their position, and felt themselves interesting subjects of inquiry. As far as he could observe they were there under a very lazy and shameful pretense of curing a disease which did not exist, by remedies which were not applied." It is unfortunate, though not unnatural, that Dr. Bucknill during his brief visit to this country, should have taken the disease dogma as represented in the so-called platform of principles promulgated by the American Association for the Cure of Inebriates as the sentiment of nearly all American physicians who had given special attention to the proper management of drunkards. And still more unfortunate that he should have taken the inebriate asylum at Binghamton, with its elegant building and grounds, and its inmates "leading for a brief time a life of indolent luxury, under a cloud of constant tobacco smoke, with cards and billiards, and only ostensible abstinence from whiskey," as the leading pattern of all, or nearly all, asylums or homes for inebriates in this country. We say it is unfortunate, first, because the doctrine thus broadly asserted that inebriety *is a disease* has been approved by only a very small part of the profession in this country, and by only a part of those who have been, and are still, connected with institutions for the reception and reform of inebriates; and, second, because the principles on which the asylum at Binghamton has been conducted, are not such as are approved by the profession at large, or by a fair proportion of those who have given special attention to the subject in this country. The provisions for retaining the inmates in luxurious idleness, broken only by exercises at cards, billiards and other games, intimately associated with drinking places and habits, is neither well calculated to cure diseases nor reform vicious habits.

Dr. Bucknill himself acknowledges that he found an entirely different system in operation in the Franklin Reformatory at Philadelphia, under the superintendency of Dr. Harris — a system which he fully approved, and expressed a wish to have introduced and fairly tried both in England and Scotland. And if he had extended his visit to Chicago he would have found there in the Washingtonian Home a much older and larger institution than the Franklin Reformatory, conducted upon the same principles, only more extended and systematical in their practical application. Instead of assenting to Dr. Bucknill's hasty decision, that American asylums for the reform of inebriates are worthless failures, we should faithfully endeavor to correct the errors and abuses thus far developed, and study more carefully the practical application of sound principles in their management. If the views we have already expressed concerning the nature and tendencies of inebriety are correct, the principles which should guide us in its management may be concisely stated as follows:

First, to remove the inebriate from the further direct action of the cause; that is, to separate him from the further direct use of alcohol or whatever intoxicant he is accustomed to use.

Second, to subject him to such intellectual and moral influences as will fully enlighten his mind in regard to the effects of alcohol on the human system, and arouse his moral faculties and conscience to a more active appreciation of his duties and responsibilities towards himself, his fellow men, and his God.

Third, to secure for him such hygienic and medical treatment as the morbid condition of his mental and physical system may need.

The whole class of drinkers or users of intoxicating agents may be divided into three groups or subdivisions, viz.: moderate drinkers, or those who do not use enough to produce intoxication, and consequently are not classed as inebriates.

These only require a faithful application of the means indicated in our second proposition, viz.: intellectual and moral enlightenment.

The second group embraces all those who have actually become so far addicted to the use of intoxicants as to become more or less frequently intoxicated, but yet are capable of exercising much self-control, and are susceptible of a strong and honest desire to be free from their vicious habit. These need, at least, temporary separation from their accustomed drinks and comrades, under such circumstances as will give them the full benefit of intellectual and moral enlightenment, with such moderate hygienic and medical treatment as will aid in removing most effectually whatever morbid effects may have been induced in their physical systems.

The third group or division embraces the whole number of those who have become confirmed drunkards, substantially incapable of self-control, and unsafe, as members of families or communities, and substantially disqualified for discharging the duties of good citizens. In these the morbid effects of the long-continued use of intoxicants on the structures and functions of the individual are such, that nothing less than a protracted isolation, accompanied by a faithful and persistent application of all the principles of management above enumerated, will afford any fair prospect of recovery.

In rendering the foregoing views available in practical application, it is evident that we need two classes of institutions or asylums for our end—one, that are especially adapted to the accommodation of those belonging to our second group of cases. These institutions should be furnished with pleasant and comfortable accommodations, and all the means for intellectual, moral, social and medical treatment, all to be administered in the spirit of genuine kindness and sympathy. Admission to, and stay in these, should be entirely voluntary on the part of the patient and his friends, subject to such rules as are necessary to ensure proper order and discipline on the part of all the inmates.

The second class of institutions should be especially adapted to the wants of those we have included in the third group of drinkers—the chronic or persistent inebriates. These should be established and maintained under state or municipal authority; and, in addition to the means of intellectual, moral, social and medical treatment belonging to the first class of institutions named, these should have regular, systematic and useful work provided for all the inmates. Admission to these should be through legal process, with legal authority to detain for a sufficient length of time to admit of thorough renovation and recovery. Our whole system of police management of drunkenness should be changed. Instead of repeated arrests, petty fines, and thirty, sixty or ninety-day sentences to Bridewells or prisons, in direct contact with other criminals, which all past experience has shown only aggravates the evil, the law should provide for having complaints against all of this class lodged with the judge of the county or other court of record, and such judge should be authorized on the certificate or testimony of two competent and well known physicians to commit the party to an asylum such as we have indicated, for a term not less than one nor more than five years, unless sooner discharged for good conduct and full recovery by the superintendent in charge of the institution. Under such a system of management this class of institutions could be made very nearly or quite self-supporting, and a very large proportion of those brought under their influence permanently restored to health and good citizenship. Such a system would place the legal relations and personal liberty of the confirmed inebriates on the same level with the insane, with institutions specially adapted to effect their reformation, and at the same time protect their friends, families, and the whole community from the depredations and crimes so constantly being committed by the class to which they belong.

We are satisfied from much personal experience and opportunities for observation, that the foregoing views are correct; and so far as they are allowed to govern us in our dealings with drunkenness in all its stages and varieties, in the same degree will our efforts be successful, both in reforming the vicious and in curing the diseased. In regard to the relations between intemperance and insanity, Dr. Bucknill presents nothing new, except a more careful analysis of such cases of insanity as have been attributed to the influence of alcoholic drinks, by which he lessens the ratio properly attributable directly to them, and increases the ratio properly attributable to complexity of causes. We had intended bestowing some attention to the other two small works whose titles are given at the head of this article; but neither time nor space will permit us to do so at present.

CHICAGO, April 9, 1879. N. S. D.

II.—CEREBRAL LOCALIZATIONS.

I. The Localization of Cerebral Disease. Gulstonian Lectures for 1878. By David Ferrier, M. D., F. R. C. S. New York: G. P. Putnam's Sons, 1879.

II. Charcot et Pitres: Nouvelle Contribution à l' Étude des Localisations motrices dans l' Écorce des Hémisphères du Cerveau. (*Motor localizations in the cerebral cortex.*) *Revue Mensuelle*, Nov., 1878, and Feb., 1879.

III. Lectures on the Localization of Spinal and Cerebral Diseases. By E. C. Seguin, M. D. Reprinted from *The Medical Record* for distribution to the author's friends. New York, 1878.

I. As the author states in a short preface, the present work is to supplement his volume on "the functions of the brain." The experimental results developed on animals in the former essay are to be applied in human pathology, and compared with the similar experiments furnished by chance in cerebral lesions. In order to establish the validity of inferences deduced from the observation of brain lesions in well observed cases, Ferrier puts into the proper light the arguments of the opponents of cerebral localization, especially Brown-Sequard. A certain lesion must be regarded as the cause of symptoms observed during life, if the symptoms are referable to no other cause, and if the simultaneous occurrence of lesion and symptom is sufficiently frequent to exclude mere coincidence. The lesion may, however, be the indirect cause of the symptoms, a relationship by no means rare in the case of the brain. But all faith in such a relationship would be completely overthrown by a single negative case; the description of such a case, however, should be received with great caution.

The functions of the brain are both physiological and psychological. The latter have usually been supposed to be diffusely distributed, and not localized in any one part. Extensive lesions in one hemisphere may be latent as regards mental symptoms; even a whole hemisphere may be disorganized with a like negative result. But, Ferrier demands, have the same parts in both hemispheres ever been destroyed without producing mental disturbance? Unless such an evidence is adduced, we can only conclude that one hemisphere is sufficient for mental action. Ferrier doubts even whether one-sided lesions are without effect on the mental state, a doubt fully justified by the impossibility of accurately gauging the mental condition, and even the usual indifference in this matter. If slight mental disturbances are recognized but with difficulty in man, how much more difficult must it be in the case of the lower animals? Ferrier warns also

—and with justice—against the readiness of some authors to apply, without qualification, the results of brain lesions in lower animals to the brain of man, when anatomy alone points out quantitative differences of no slight import.

The existence of motor centres in the cortex is defended by Ferrier in a very judicious manner. A false importance has been attached by many to the fact that movements can be produced by electric irritation, even after removal of the grey substance of the cortex. But is not every motor nerve excitable as well as the centre from which it originates? That, however, the motor impulse is started primarily in the grey cortex and not in the subjacent white fibres, is sufficiently proven by the greater strength of current necessary in order to stimulate directly the exposed fibres. A conclusive evidence is also furnished by the experiments of Franck and Pitres, who found the interval of time between the stimulus and the movement notably lessened by removal of the grey substance.

Although there seems to be an apparent want of harmony in the results of localized destruction of the cortex in different animals, the entire results come readily under one principle, if we remember the relative development of the cortex and the lower centres in the different species. The principle could not be better expressed, than in Ferrier's words: "In accordance with this generalization, I ventured to predict that, even in the case of animals whose motor powers did not seem permanently to suffer from destructive lesions of the cortical motor centres, those movements must be paralyzed which involve conscious discrimination, and were not automatically organized. This has been amply verified by Goltz's experiments on dogs. Goltz found that, though a dog's paw is not permanently paralyzed as an organ of locomotion by destruction of the cortex, *yet it remains permanently paralyzed for all those actions in which it is employed as a hand.*"

Entering now upon his special subject, Ferrier quotes a number of cases of injury to, and lesions in, the frontal lobes. In these cases, collected from different sources, no motor or sensory symptom can be legitimately traced to a lesion of the frontal lobes; psychic troubles are, however, mentioned in some. Mental disturbances Ferrier would expect in every case, strictly examined, a result which would harmonize with his experiments on the præ-frontal lobes of monkeys. These animals, "while not actually deprived of intelligence, had lost, to all appearance, the faculty of attentive and intelligent observation."

Hereupon, lesions of the motor regions are discussed, under the headings of hemiplegias and monoplegias, and on the other hand, of general unilateral spasms and (limited) monospasm. An unbiased attempt is made to classify the lesions observed in the cases topographically, and the result agrees very well with the author's experiments on monkeys. We cannot here follow the author in his details.

Less preciseness is to be found in the chapter on lesions of the sensory region. Anatomically, the occipital and temporal lobes are connected with sensory strands. The experiments of Veyssière (repeated by other French observers) show the functions of the strands, their section at the level of the posterior third of the internal capsule producing hemianæsthesia of the opposite side of the body. Lesions situated in this part of the human brain have the same result.

Ferrier's experiments on monkeys lead him to regard the occipital lobes as the centres for visual sensations. He admits, however, the precarious nature of this view. A number of cases, both of slow lesions and of sudden accidents of these lobes in man were wholly negative in symptoms. Munk's positive statements, however, as regards the connection of the eyes with the occipital lobes, are dismissed in a somewhat unsatisfactory manner. Recent clinical observations, such as those of Baumgarten and Huguenin, point decidedly towards the existence of visual centres in the occipital lobe.

The parieto-temporal region is considered by Ferrier to contain the centres of the senses. His irritation experiments, however, on monkeys afford but a very feeble evidence for this view. But it must be admitted that such uncertainty is unavoidable in irritations of sensory parts in animals — especially nerves of special sense. On the other hand, the experimental destruction of these centres is followed by symptoms, the interpretation of which as paralyses of special senses seems natural. Strangely, however, a number of clinical cases from high authorities were absolutely latent as regards sensory symptoms connected with lesions of the parieto-temporal region. No bilateral lesions, however, have been reported without sufficient mental disturbance to render sensory examinations valueless. To overcome the apparent discrepancy between the physiological and pathological observations, Ferrier cuts, instead of untying, the Gordian knot, by claiming that such cases have been *loosely observed.* Although this charge is not without some foundation, it does not throw any further light upon the subject. Our entire knowledge of sensory centres in the cortex is as yet very fragmentary, and if Ferrier undertakes the rôle of pioneer in this field, we must permit him to take some stand-point to start from. Aside from the chapter on sensory centres, the author cannot be accused of theorizing without proper basis. On the contrary, the entire work is a very judicious and critical review of facts.

II. Charcot and Pitres published about a year ago a long series of articles (also in the *Revue Mensuelle*) on the subject of cerebral localization. Their present memoir is but an addition to the previous one. It consists almost wholly of reports of cases, many of which are original. Apparently adverse observations are impartially criticized, while the others, selected with due care, in order to exclude complications, are analyzed. The value of the memoir is, perhaps, best expressed in the author's own conclusions.

There exist in the cerebral cortex regions which possess no motor functions, and can be destroyed without motor impairment. These regions comprise the occipital, the sphenoidal (temporal), the anterior part of the frontal, the orbital and the parietal lobe (excepting, perhaps, the foot of the latter), also the lobus quadratus and cuneatus.

The centre for the movements of the tongue is found at the foot of the third (inferior) frontal convolution, and on the contiguous portion of the ascending frontal convolution.

The centre for the movements of the inferior part of the face occupies the lower part of the two ascending (frontal and parietal) convolutions.

The centre for isolated movements of the superior extremity exists in the middle third of the ascending frontal convolution.

The combined centre for movements of the upper and lower extremity (of the opposite side) is spread over the paracentral lobule, the superior third of the ascending frontal and the upper two-thirds of the ascending parietal convolution.

III. In a series of eight lectures delivered at the College of Physicians and Surgeons, Dr. Seguin attempts to facilitate localized diagnoses in nervous diseases, by giving a short, but comprehensive, sketch of the physiological anatomy of the cord and brain, including the most recent researches. As far as known, the functional importance of the different strands is likewise stated; and thus there is developed a schema, from which lesions can be readily localized. As such, it has some of the faults, but also all of the merits, of a schema. It can certainly serve as an excellent guide in the labyrinthine arrangement of the nervous system. The diagrams that are given are very useful in illustrating the text, though that is itself sufficiently clear, and the critical remarks of the author are, throughout, judicious. It is to be regretted that so useful and convenient a condensed statement of its subject is not in a form more generally accessible to the medical student. H. G.

SHORTER NOTICES.

I. Health, and How to Promote it. By Richard McSherry, M. D. New York: D. Appleton & Co., 1879. 185 pages. Chicago: Jansen, McClurg & Co.

II. Physiology: Preliminary Course Lectures. By James T. Whittaker, M. D. Cincinnati: Chancy R. Murray, 1879. From Robt. Clarke & Co.

III. On Rest and Pain: A Course of Lectures on the Influence of Mechanical and Physiological Rest in the Treatment of Accidents and Surgical Diseases, and the Diagnostic Value of Pain, delivered at the Royal College of Surgeons of England in the years 1860, 1861 and 1862. By John Hilton, F. R. S., F. R. C. S.; Edited by W. H. A. Jacobson, F. R. C. S. Second Edition. New York: William Wood & Co., 1879. Chicago: Jansen, McClurg & Co.

IV. A Practical Manual of the Diseases of Children, with a Formulary, by Edward Ellis, M. D. Third edition. New York: William Wood & Co., 1879.

V. Health Primers: No. 1.—Exercise and Training; No. 3.—The House and its Surroundings; No. 4.—Premature Death, Its Promotion or Prevention. New York: D. Appleton & Co., 1879. Chicago: Jansen, McClurg & Co.

VI. Essentials of Chemistry—Inorganic and Organic. For the Use of Students in Medicine. By R. A. Witthaus, A. M., M. D. New York: William Wood & Co., 1879. Chicago: Jansen, McClurg & Co.

VII. On Fracture of the Femur. By Edward Borck, M. D. With Illustrations. St. Louis: G. G. Rumbold & Co., 1879.

VIII. New and Original Theories of the Great Physical Forces. By Henry Raymond Rogers, M. D. Published for the Author. New York, 1878.

IX. The Non-Asylum Treatment of the Insane. By W. A. Hammond, M. D. G. P. Putnam's Sons, New York, 1879.

X. Notes on the Treatment of Skin Diseases. By Robert Liveing, A. M., M. D. Fourth Edition, Revised and enlarged. New York: Wm. Wood & Co., 1878. Chicago: Jansen, McClurg & Co.

XI. Atlas of Skin Diseases. By Louis A. Duhring, M. D. Part V. Philadelphia: J. B. Lippincott & Co., 1878. Chicago: Jansen, McClurg & Co.

XII. METALLOSKOPIE UND METALLOTHERAPIE. Von Prof. Dr. Eulenburg in Greifswald. (Reprint from the *Wiener mediz. Presse.*)

I. As a general rule we are not unqualifiedly prepossessed with the average work or publication on personal hygiene: there is too much of a certain kind of quackery associated in our mind with this kind of literature. The medical popularizer is so often only the advertiser of a nostrum or a system, that his credit with the regular profession is not, perhaps, first-class — nor does it always deserve to be. But these remarks, in a notice of the present work, are made only for the purpose of saying that it is not to come under the ban of any *a priori* condemnation; that it belongs to a class of works that, so far as they go, may do much good to their readers, the laity, to whom they are especially addressed. It is a very legitimate contribution to popular sanitary literature, and not in any proper sense a medical book. The author reviews in a rapid way the various periods of life, and the dangers that beset them, and makes it a sort of general hygienic guide-book, a manual of health for the individual. It does not go fully into details, it follows in this the safer course; but its advice is good, and stated in an attractive and readable manner. The physician ought not to require the information this volume contains; but it is one that he can safely recommend to his patients and friends. It is neatly and handsomely gotten up.

II. This volume consists of some twelve lectures delivered at the Medical College of Ohio by the author, who holds the chair of physiology and clinical medicine in that institution. Though thus few in number, they are sufficiently comprehensive in their subjects, since they range over the whole field of biology, paleontology and evolution, as well as some of the more purely physiological themes. Dr. Whittaker is fully in accord with the most advanced theories, and accepts them with somewhat less reserve than we should ourselves. An evidence of his tendencies is shown in some of his references, such as that to Carl Vogt, where he calls him "one of the most profound philosophers of his day." We cannot, however, justly disparage in any degree his book ; it contains nothing that a medical man ought not to know, but we are of the opinion that few obtain all its information during their undergraduate course in the lecture-room. As an introduction to the study of physiology it is the most comprehensive we have yet seen in so small a compass, and one of the most attractively written. Dr. Whittaker is evidently well up in his subject, and if the following lectures in the regular course are in any way as full and valuable as these, the students at the Ohio Medical College are especially favored—provided, always, that their preparatory training has been equal to the demands of this preliminary course. The book is well worth reading, not only by the medical student, but also by the practitioner and the cultivated layman.

III. This is the republication of the second edition of an English work of considerable value. It consists mainly of a discussion of numerous clinical histories illustrating the effects of mechanical and physiological rest in the treatment of various surgical and other disorders, many of them of decided interest, though they are hardly, in all cases, discussed with reference to the latest information which we have on the physiology of the nervous system. It is a book that can often be consulted with profit by the practitioner, and contains a great deal of plain common sense applied to medical and surgical treatment. The wood-cuts, which, though not artistic, illustrate the text very fairly, and the typography, etc., are all that can be demanded for the very reasonable price at which this series is sold — one dollar a volume.

IV. This, the second volume of Wm. Wood & Co.'s dollar series of medical books, is, like its predecessor, an American republication of an English work. It belongs to the general class of condensed manuals — almost too condensed, in fact, to be the sole text-book for the student in its department. Still, it may serve a very good purpose as a work of reference for the busy physician; for his purpose the quality of the contents of the work in some respects may atone for its quantitative deficiencies, which he can supply out of his own knowledge. And yet there are many features in this work which we like; among them we may mention its cosmopolitan character: the author having left Great Britain for one of its colonial dependencies is less limitedly insular or European in his references, and, presumedly, in his reading, than are many of the Transatlantic writers. Though it is of no special advantage to an American physician to see his own countrymen freely quoted by a foreign writer, yet it is not so common as to be unpleasing to him. The remarks on treatment, also, are generally very sensible and practical.

Among its deficiencies we notice nothing is said in regard to cerebro-spinal meningitis, cholera infantum as a special variety of gastro-intestinal disorder, and of various surgical and congenital conditions in children that are frequently met with and must be attended to by the practitioner. What is given is generally so well stated that the regret for the omissions is increased. The therapeutic hints and formulary at the end of the work will undoubtedly be of use to many of its readers.

In conclusion we can honestly recommend the book, not as a comprehensive and complete treatise on the diseases of children, but as, what the author states was his intention to make it, a handy book of reference to the student and practitioner.

V. These little volumes are not intended for the physician, but for the general public, to whom they ought to be of much value.

Each primer is written by a medical man specially fitted to speak upon the subject ; the list of editors and contributors, including as it does such names as Crichton-Browne, Tilbury Fox, Sidney Ringer, Henry Power, and others, is such as to insure confidence that the little books are none of them open to the objections we have already alluded to as valid in the case of many popular sanitary publications. The American editions are neatly gotten up, and furnished at a price (forty cents) well within their value and the reach of every one to whom they are addressed. We shall notice some of them upon special subjects in other places, as they appear from time to time, and would bespeak for them the largest circulation.

VI. This little book is a convenient pocket hand-book of the principal medical essential points of chemistry, arranged in the form of a catechism of questions and answers. For those who wish to have always at hand for convenient reference a memorandum of chemical compatibilities and incompatibilities of drugs, and medical chemical tests, it will prove very useful. Its form and arrangement would, we should think, preclude its ever being used as a text-book, or even a convenience for "cramming" for examinations ; it is hardly complete enough for either of these uses, and therefore is almost or quite unobjectionable in those respects. In size it is hardly larger than the average pocket dose book, and can be conveniently carried in a medicine case if so desired.

VII. Dr. Borck's little treatise on Fracture of the Femur consists of two parts: the first describes the author's own method, and the second is a general review of the treatment of this accident. Dr. Borck's method consists in the use of a double inclined plane, with traction on the thigh ; and the memoir as a whole may be considered as an argument in favor of this plan. It is issued to meet the demand for reprints of his original articles in the *St. Louis Medical and Surgical Journal*, which form the substance of the volume.

VIII. New Theories of the Great Physical Forces is a rather ambitious attempt to account for the phenomena of the universe in a manner altogether different from that which has been accepted by the scientific world generally. It is Dr. Rogers' idea that all the forces of nature are really one, which he calls magnetism, or, rather, vito-magnetism; and from this stand-point he attempts to account for all physical phenomena. The book is really only a statement of his views — not an argument in their favor; and as he or any one else has the privilege of holding them at pleasure, perhaps no objection can be made to it. It will not convince any one except such as choose to follow the author's authority. It is not possible for scientific men to take reasonable offense at the temper of the work ; the apology in the preface should insure against such a feeling.

IX. Dr. Hammond's paper, read before the Medical Society of the State of New York last February, is a plea for the better understanding on the part of the people in regard to insanity, and a protest against the exclusive claim made by asylum superintendents that they are the only ones who understand the subject and are entitled to speak upon it *ex cathedra*, and that this wisdom will die with them. He shows that the present condition of public sentiment in regard to the care of the insane, and which has been largely brought about through the efforts of asylum physicians, is to a great extent erroneous, and that it tends in many ways to the public disadvantage. The exclusive treatment of the insane in large asylums leads to great public expenditures; the tendency of these asylums, under their present system of management, is too often in a direction altogether opposed to the most scientific and effective treatment of the disease. We shall have frequent occasion to allude to this subject. The question of asylum reform is one to which this journal is fully committed, and may include this paper elsewhere in a more extended notice. We commend it to the attention of thoughtful members of the medical profession.

X. This is a new edition of this very convenient abbreviated hand-book of skin diseases. The changes and additions are slight, being mainly in the remarks on classification, and a few other places. The index is also a useful addition, even in so small a volume as this.

XI. The fifth number of Dr. Duhring's atlas of skin diseases is fully up to its predecessors in artistic and literary merit. The affections described and figured are scabies, herpes zoster, tinea sycosis and eczema (vesiculosum). The only general remark we will make about it is, that it appears to us to be the finest and most useful work of its kind that we have ever seen.

XII. In this very critical article Dr. Eulenburg reviews the different statements and reports on metalloscopy, chiefly by French observers. His own experiments on the galvanic currents produced by contact of metals with the human skin are detailed; but he admits that they afford no clue for the solution of the different problems. On the whole, he cautions against both unjust scepticism and hasty belief, occupying himself a stand-point of critical expectation.

Editorial Department.

MANAGEMENT OF THE INSANE.

THERE have been, since our latest references to this subject in the pages of the JOURNAL, numerous fresh evidences brought to light that there is need for a careful examination by duly constituted authorities, and based on this a reform of the actual practical management, in general and in detail, of our asylums for the insane. Under this head we would call the attention of our readers to the movement set on foot in the State of New York, having as its object a thorough examination into the past and present condition of the numerous asylums within the limits of that great commonwealth. It is in the form of a petition addressed to the New York Legislature, numerously signed, and a copy of which we subjoin. It is as follows:

"*To the Legislature of the State of New York:*

"The Petition of the undersigned Physicians, Lawyers, and other Citizens of the State of New York, represents as follows:

"There has lately sprung up a general and marked discontent in the public mind with regard to the management of our insane asylums. From the nature of the case, the internal mechanism of these institutions being more or less secluded from public scrutiny, it was not to be expected that the popular feeling could point to any special fault in the system as its cause. Like most popular movements, the agitation of the asylum question has, until quite recently, rested on isolated and flagrant instances of abuse, rather than on the great systemic defects of which these instances were but the outcome.

"But within the past few years, members of the medical profession, whose studies led them to investigate asylums, without bias or prejudice, purely in the interests of medical psychology and philanthropy, have made public charges against the system of asylum supervision and asylum management in this State which are deserving of notice.

"They merit special attention for the single reason, if there were no other reasons, that those now at the head of our asylums, have been either unable or unwilling to answer their arguments, or to disprove their allegations.

"Many of these defects, medical and administrative, have been admirably and fully set forth by Dr. H. B. Wilbur in his 'Report on the Management of the Insane in Great Britain,' published in 1876 and 1877. This writer institutes a comparison between the management of the insane in the two countries which is as humiliating as it is instructive.

"The following points are among the most prominent features of the pending inquiry. Some of them rest upon exact evidence, others can be substantiated by responsible members of the medical profession, and regarding still others, strong circumstantial evidence can be adduced. It is the purpose of the projected investigation to discern how many of the questions herein set forth can be satisfactorily answered under oath. We also believe that the points referred to by these queries are of the highest importance for the welfare of the unfortunate insane.

"QUESTIONS RELATING TO THE MANAGEMENT OF ASYLUMS.

"1. How many days in the year is the Superintendent actually and really on duty in his office or in the wards?

"2. How many days in the year is the Superintendent away from his asylum engaged in private business, in medical or medico-legal consultations, attending upon courts without an order from the Attorney-General or other proper authority?

"3. Does not the State (or city), in giving the Superintendent a fair or large salary, make a contract for the use of his whole time, usual vacation excepted?

"4. How often in a week does the Superintendent see and speak to *every* patient under his charge?

"5. How many patients in the asylum are seen only once a month, or less often, by the Superintendent?

"6. How frequently does the Superintendent make unexpected visits to his wards? How often does he make tours between midnight and morning, to observe the symptoms of some patients, and to see how nurses and watchmen perform their duties?

"7. How many patients are actually and really under the Superintendent's own medical treatment?

"8. Does the Superintendent see and examine every new case immediately after admission; and is he responsible for the registered diagnosis, and general treatment?

"9. Does the asylum possess, and do its medical officers really use such common instruments as the thermometer, ophthalmoscope, æsthesiometer, dynamometer, sphygmograph, microscope, faradic and galvanic electrical batteries, the speculum and uterine sound, chemicals for the analysis of urine, etc.?

"10. Is feeding by force *always* done by a medical officer or in his presence, as it should be?

"11. Is the Superintendent consulted whenever forcible restraint is required; and is the duration, manner and result of this mechanical restraint duly recorded in a book kept for the inspection of the Commissioner in Lunacy and of the courts?

"12. How frequently does the Superintendent avail himself, for the benefit of his patients, of consultations with general physicians and surgeons, and gentlemen eminent in the several departments of medicine?

"13. Do you ever employ the barbarous and injurious means of restraint known as a 'crib?'

"14. Are undergraduates in medicine ever employed in your asylum as assistant physicians, or acting assistant physicians?

"QUESTIONS REFERRING TO THE INSPECTION OF ASYLUMS.

"1. Does the Commissioner in Lunacy give any notice of his coming to the officials of an asylum he is about to inspect?

"2. Has the Commissioner ever visited asylums at unusual hours, as in the middle of the night or in the very early morning, in order to determine the usual condition of their wards?

"3. Does the Commissioner ever go about an institution without company, or at any rate without a medical or other officer whose presence can prevent free speaking on the part of attendants and patients?

"4. How frequently are inspections made in the various asylums; and how many days in the year is the Commissioner in Lunacy engaged in private business not legitimately appertaining to his office?

"5. Does not the State, in paying the Commissioner a fair salary, make a contract for his whole time, customary vacation excepted?

"6. Was the present Commissioner appointed in strict accordance with the law which requires the candidate for this position to have been a 'physician of experience?'

"Besides, your petitioners believe the following statements to be well founded. They refer to more strictly medical matters than the above-recited questions, yet we consider that if these evils exist, the State and the patients in our asylums are highly interested in their eradication.

"1. Superintendents of insane asylums are, nearly without exception, not chosen from among medical men who have pursued special studies in neurology at home and abroad, and who are well trained physicians, but from among assistant physicians of asylums who, after having been badly chosen (*vide infra*), have passed a number of years immured in an institution.

"2. Assistant physicians of asylums (future candidates for the position of superintendent) are nearly always men just issued from our too elementary medical schools; men who have not served in civil hospitals (which can be entered only by severe competitive examination); their qualifications are not submitted to any test; when in the institution they are not furnished with means of study (medical journals, books and instruments); and, inevitably, as years go by, they forget what general medicine they knew on graduating.

"3. Assistant physicians, moreover, are overworked, and wretchedly paid. Their time is taken up by visiting too many patients, by writing interminable, useless histories of cases, and by various 'official' duties, such as talking by the hour with friends of patients, receiving visitors, etc. The largest asylum under State management (in this State) has only four assistant physicians for between six and seven hundred patients. In a general hospital, like Bellevue or the New York Hospital, every division of from forty to eighty patients is officered by one attending physician (who really sees his patients daily) and three assistants, all graduates, selected from among a crowd of the best men by a severe competitive examination. And it must be borne in mind that in general hospitals there are, as in insane asylums, very many chronic cases requiring a minimum of care. From this comparative statement it is at once evident that in spite of enormous outlays of money our insane asylums are indifferently officered.

"4. Superintendents and their assistants, with hardly an exception, are not versed in the new anatomy and physiology of the nervous system, the part chiefly concerned in insanity.

"5. Superintendents and their assistants, with hardly an exception, are not believed to be skilled in the modern methods of diagnosis and of post-mortem examination. Few of them are able to read in the original the invaluable contributions to insanity and its treatment which we owe to German and French scientific physicians for the insane.

"6. The little pathological work which has been done in our asylums at enormous cost has been of the most elementary sort, and has been ridiculed at home and abroad. With the liberal aid it receives from the State, the pathological laboratory in one of our asylums did not furnish the materials for successful competition for the great Tuke prize, for the best essay on the pathological anatomy of insanity, offered in England last year.

"7. In some of our asylums the pernicious practice of allowing undergraduates, accepted without *bona fidė* examination, to act as assistant physicians, is tolerated. This is highly unjust to the patients, who have a right to medical attendance in the legal sense, and also to practitioners outside. The lunacy laws of 1874 provide that no practitioner can certify to the lunacy of a patient unless he have been three years in the practice of his profession; and it thus happens that this experienced physician's diagnosis and certificate are in a measure subjected to the revision and control of men who have not yet obtained their degree, or of others who have just passed from the benches of the medical school to the asylum.

"In view of the above numerous reasons for believing that there exists gross mismanagement in the medical administration of insane asylums in this State, your petitioners do respectfully request that your honorable body appoint a committee for the examination of the management of all institutions for the care of the insane in the State of New York.

"And your petitioners will always pray, etc."

These are serious statements, and it is sincerely to be hoped, whether true or not, they will be once for all taken up by the proper authorities, and the fullest opportunity given, not only to establish, but, if it can be done, to refute them. Until this is done, asylum management in this country must and will remain under painful suspicions, whether fairly or not. Various recent occurrences have only served to increase these suspicions in the public mind, whether professional or general. We would cite as instances the results arrived at in a Minnesota asylum — at one in particular in the State of Ohio, and at others, such as in Michigan, New York, Massachusetts, etc. Examples of the most grave faults of management, financial, administrative and medical, have been recently made known, which give serious emphasis to such a memorial as the one quoted above, and the particulars relating to many of which are accessible to us, but for which we have no room at present. But, in view of what is already known, we feel no hesitation in joining in the demand for a thorough investigation of the whole subject of asylum management in this country, and for devising some better means than now exist for detecting and remedying shortcomings and abuses of whatever kind. It should result in benefit, not only to such asylums as are notoriously mismanaged, but even to such as are practically above reproach.

We earnestly hope that the same earnest and intelligent effort may be made in other States as the above petition shows is being made in the State of New York: to place asylum management on a better footing, from every point of view. We wish to see less of the one-man power in the asylums, and a more efficient and responsible system of inspection and control.

We cannot, in our editorial columns, adequately notice the decease of every noted person in neurological medicine,—our space is not sufficient for that purpose; but, since our last issue, the news has come of the death of two men so worthy of special notice, for such different reasons, that we could well afford to break a custom that we have followed in the main for the past two or three years. In the case of Dr. Benedict Stilling it is only justice to do honor to his memory, for his works have had but scanty recognition as compared with their merits during his life; and though Prof. Tardieu was not, strictly speaking, a neurologist, his name is, nevertheless, so great a one in the literature of insanity, that his death cannot be passed unnoticed by us. Though known to some extent as a writer on pathological subjects, his great reputation was made in the departments of forensic and public medicine; and in this his abilities received ample recognition. It is probable that no one man was ever as much an authority in medico-legal matters before the French courts as Prof. Tardieu. The number of his separate reports on such subjects as were referred to him as expert by the tribunals had reached in 1876, when his active life may be said to have ceased, to the almost incredible number (according to Prof. Chaufford) of five thousand two hundred and thirty-nine.

Dr. Stilling was an entirely different type of an investigator from the able and brilliant Parisian professor. A practicing physician and surgeon in the little city of Cassel, he was scarcely known even to his own countrymen — or, rather, perhaps his reputation was but a vague and general one to the many — and he was properly appreciated only by the few. And yet he was one of the most accurate and extensive investigators of the microscopical anatomy of the nervous system that has ever lived; and even his earlier works at this day are marvels of research.

His first important neurological publication, to our knowledge, was a work of several hundred pages on spinal irritation, printed in 1840. In 1842, in conjunction with another physician of Cassel, Wallach, he published his first researches on the spinal cord. In 1843 appeared his treatise on the medulla oblongata, followed in 1846 by his great work on the pons varolii, printed in Latin and German, which was specially mentioned by Schrœder Van der Kolk as a remarkable monument of German industry and perseverance, while at the same time he noticed the neglect it had received from anatomists. These were followed by his still more extensive and valuable newer researches on the spinal cord, published in 1856–1859, and his similar work on the cerebellum, the first part of which made its appearance in 1870, and which has just been completed. It is difficult to characterize these works, we know scarcely anything that is at all comparable to them in many respects, considering the time of their appearance, and the other circumstances of their production.

Besides these Dr. Stilling was the author of various contributions to the medical periodical literature of his country, some of them within the past two or three years. All this literary work was done in the leisure time of a general practitioner and surgeon; he never, so far as we know, held any professional or other position especially favorable to such extended personal scientific research.

One reason for the fact that the labors of Dr. Stilling received so little recognition, at least until lately, is perhaps that which has already been suggested by Schrœder Van der Kolk, viz., the magnitude and form of his publications has deterred anatomists from their examination. It is not a creditable showing that such a mine of valuable observations should have been neglected on account of its richness and extent. Perhaps a better reason is to be found in the fact, that while Dr. Stilling was pre-eminent as an observer, he was not equally great in making deductions from his observations, he made no startling and brilliant generalizations to call attention to his work, and it may be was sometimes wrong in those that he did make. His works have been, therefore, almost a *terra incognita* to the physiological student, notwithstanding that they contain facts that are full of suggestiveness to him.

It was apparently Dr. Stilling's intention to complete the anatomy of the central nervous system in a systematic and thorough manner, and it is to be hoped that the part on the cerebrum, the only one lacking, has been left in such a form as to admit of its publication under proper supervision. But whether this be the case or not, enough has been done to make him remembered as an investigator, who, without notable advantages, was one of the most accurate and indefatigable that the world has ever known in this department; who apparently caring little for present reputation, continued to do honest and faithful work, until long after the usual period of active literary production.

In this connection we should also notice the recent death of Dr. Isaac Hays, of Philadelphia, the oldest medical editor, and indeed the oldest living editor in the United States, the news of which has just reached us as we go to press. He had been editorially connected with the *American Journal of Medical Sciences* almost or quite from its beginning, and to him is largely due the high character it has attained at the head of American medical journalism.

Since our last issue, the death of Dr. John M. Woodworth, Surgeon General of the United States Marine Hospital Service, has occurred. Dr. Woodworth had succeeded in bringing the service over which he presided to a high state of efficiency. But few knew, or ever will know, of the amount of self-sacrificing, disinterested labor he expended in the work he accomplished. He has left behind him a rare reputation as an executive officer, and for official integrity.

We have received the first number of Dr. Seguin's new bi-monthly journal, the *Archives of Medicine*, published by Messrs. G. P. Putnam's Sons, New York. This is to succeed the series of American Clinical Lectures, which has just been discontinued, and is also to be considered as a continuation of the *Archives of Scientific and Practical Medicine*, edited some years since by Dr. Brown-Sequard and the present editor. The character of the *Archives* is insured by the name of the editor, and the first issue fully meets all requirements. Its make-up, also, is one of the handsomest we have seen.

STEVENS' TRIENNIAL PRIZE, 1882.—This prize, established by Alexander H. Stevens, M. D., amounts to two hundred dollars. The subjects for the next prize are as follows:

I. *Lesions of the brain, in connection with the two forms of diabetes.*

II. *Diphtheria, in its relations to membranous croup.*

The competing essays, on either of the above subjects, should give an account of our present knowledge and also the results of personal investigation. They must be transmitted to the President of the College of Physicians and Surgeons, New York, on or before the first day of January, 1882. Each essay must be designated by a device or motto, and must be accompanied by a sealed envelope, bearing the same device or motto, and containing the name and address of the author. The envelope belonging to the successful essay will be opened, and the name of the author announced at the annual commencement of the above-named College, in March, 1882.

This prize is open for universal competition.

J. C. DALTON, M. D.,
Secretary of the Commission.

WITH the present month Dr. W. A. Hammond, one of our associate editors, announces a new neurological quarterly devoted entirely to the publication of original articles, and entitled *Neurological Contributions*.

On account of the slowness and difficulty in getting authors' proofs of articles, and some other causes, the Journal has not appeared on time. Such delay in its appearance must seldom occur.

On account of the press of matter into the first department of the Journal we have been obliged to omit several reviews until the next issue.

Periscope.

a.—ANATOMY AND PHYSIOLOGY OF THE NERVOUS SYSTEM.

THE PSYCHO-MOTOR CENTRES OF NEW-BORN ANIMALS.—The researches by Prof. J. Tarchanoff (*Revue Mensuelle*, Oct. and Nov., 1878,) were intended to examine further the interesting fact found by Soltmann, that the cortical motor centres are not excitable in the new-born dog until the tenth day, from which time on they successively develop in function. Soltmann concluded that all movements of the new-born animal are purely reflex, and in this view he was confirmed by the absence of all symptoms on destruction of the hemispheres before the tenth day. Subsequently this author found further that at this early period no inhibitory influence on reflex action was exerted by the hemispheres, and that the inhibitory action of the vagus on the heart was also wanting.

Soltmann experimented only on puppies and rabbits, animals which are born blind.

Tarchanoft, however, compared these results with experiments on new-born guinea-pigs. This animal, born with open eyes, is also more developed at birth, as is shown by his bony skull and the existence of furrows (four in number) in the cerebral cortex. In the new-born guinea-pig Tarchanoff could easily excite isolated muscular movements by faradizing the cortical centres. The function of the centres could even be demonstrated on the fœtus of guinea-pigs some days before birth.

The inhibitory influence exerted by the cortex on reflex actions could also be demonstrated in the new-born guinea-pig, by measuring the time a current passing through the hind leg required in order to produce a reflex movement. This time was very considerably lengthened by electric or mechanical irritation of the cortex.

In the new-born guinea-pig the inhibitory action of the vagus on the heart was also marked in Tarchanoff's experiments.

Previous researches by Bibra and by Schlossberger have shown that the fœtal brain contains more water, but less fats and phosphorus than the brain of the adult. It has also been shown that the grey substance contains more phosphorus than the white in the adult, but not in the fœtus.

Repeating these observations, Tarchanoff found less difference between the brain of the adult and the new-born in the guinea-pig, than in either dog or rabbit. For microscopic purposes, the brains were hardened in Müller's fluid; the brain of the new-born guinea-pig was well hardened after four weeks, that of the dog or rabbit at birth required six to eight weeks.

In the brain of the new-born rabbit the cortical grey substance is still in the embryonic state; there is but an imperfect differentiation of the cells, and the subsequent pyramidal shape of the ganglionic cells can scarcely be recognized. The nerve fibres are entirely without myeline.

In the guinea-pig, on the other hand, at the same time of life, the grey substance is almost, though not quite, completely developed, there being still some embryonic cells. The fibres, however, are all medullated.

In order to find some anatomical reason for the difference in the degree of inhibitory influence exerted by the vagus on the heart of adults and fœti, Tarchanoff measured these nerve fibres microscopically. In the new-born rabbit the vagus has but little action on the heart, and its fibres are still very thin. The fibres in the adult are two to three times as thick, and have a medullary sheath five to twenty times as thick as the vagus fibres in the new-born rabbit. The functional power of the nerve increases directly with the anatomical development of its fibres. The want of activity of the fœtal vagus is not due to imperfect development of the inhibitory ganglion in the sinus venosus, since Tarchanoff, differing in this respect from Soltmann, could arrest the heart by exciting the sinus venosus directly.

In the young guinea-pig the vagus is nearly as active as in the adult animal, and accordingly Tarchanoff found but a slight difference between the size and the development of the fibres of the fœtal and adult guinea-pigs.

In order to test his hypothesis, that the action of the vagus depends on the development of its fibres, and especially their medullary sheath, Tarchanoff examined the vagus of the pigeon, in which animal the nerve has but little power over the heart. The fibres were found very thin, and possessing but a feeble minute myeline covering as compared with other nerves of the bird.

As a general fact quite worthy of notice, the author found that the effect of stimulation of *one* vagus is always increased by dividing the vagus of the other side.

In order to study the development of the nervous system under different influences, Tarchanoff cites hereupon a large number of experiments made on litters of puppies, rabbits and guinea-pigs. The animals of one litter were kept in the same conditions in general, but subjected to different experimental influences. Some received daily a few spoonfuls of alcohol, others a small quantity of phosphorus (about $\frac{1}{80}$ grain), dissolved in cod liver oil. Some were put from one to two hours a day into a condition of cerebral hyperæmia by being suspended with the head downward. The brain of others was, on the other hand, rendered anæmic by placing the rear part of the body into a partial vacuum for some hours each day. The influence of the hyperæmia thus produced in the posterior part of the body was at once evident on inspecting the exposed brain. As soon as the air was exhausted from the box in which the animal's hind legs were included, the brain became anæmic and less excitable.

As a result of extended observations, Tarchanoff states that the administration of phosphorus, as well as the repeated production of cerebral hyperæmia, results in a hastening of the opening of the lids, and an increase of excitability of both psychomotor and reflex centres. There is all evidence of a superior development of the nervous system by the use of phosphorus, or production of repeated hyperæmia. Besides, the brain of the animals so treated contains more than the normal amount (at that age) of solid constituents and phosphorus.

The very opposite effects are produced by daily doses of alcohol or the frequent production of cerebral anæmia.

As incidental results, the author mentions further that there is a difference between the two hemispheres in new-born animals. The centres for movements of the limbs are more excitable in the *left* hemisphere, while mastication can be provoked more easily from the right hemisphere. This observation, if applicable to man, would explain right-handedness.

There is a certain correlation between the activity of different cortical centres. The natural act of mastication will, for instance, reduce the excitability of the centres governing the limbs. The cortical excitability is also much reduced by sleep and by derivation of attention, for instance by the sight of meat (young puppies). The same influences diminish also the reflex excitability of the animal. H. G.

SECRETORY AND TROPHIC GLAND NERVES. — R. Heidenhain, *Pflüger's Archiv*, XVIII., p. 1. From his researches on the submaxillary gland of the dog, H. formulates the law, that the *percentage* of salts in the saliva increases with the total amount of saliva, when the secretory nerve (chorda) is irritated up to a certain limit. This increase of salts occurs independent of the physiological condition of the gland. The amount of *organic* solids, however, depends not only on the degree of irritation of the nerve, but also on the state of rest or fatigue of the gland. With the same degree of excitation of the secretory nerve, the percentage of organic solids diminishes the longer the gland has been in action. By increasing the excitation of the nerve, the proportion of organic residue augments likewise. But if the secretory nerve of a fresh (not fatigued) gland be stimulated, the percentage of organic matter will remain high after cessation of the stimulus, and diminish subsequently less rapidly than the inorganic salts. Hence, H. concludes that the secretion of water and salts on the one hand, and of the organic substance on the other hand, occurs under different conditions.

As regards the parotid gland of the dog, H. confirmed the statement of Loeb, that the secretory nerve is a branch of the glosso-pharyngeal nerve, entering the tympanic cavity with the tympanic branch, and passing thence into the cranial cavity to join the superficial pretrosal nerve. It finally reaches the gland with the auriculo-temporal branch of the trigeminus. The other gland-nerve is the sympathetic. The latter contracts the blood-vessels of the gland; the former dilates them. The pressure under which the parotid secretion can still occur, may amount to a column of mercury of 106 to 118 mm. in height, a tension greater than the blood pressure in the smaller vessels.

After prolonged action of the gland, the parotid saliva becomes impoverished in organic constituents ; less so in the amount of salts. By increasing the electric stimulus, both organic and inorganic constituents increase in percentage; but if the gland is exhausted, only the *salts* augment in proportion.

Heidenhain could not find any influence of the sympathetic on the amount of parotid saliva. But if the two secretory nerves (cerebral and sympathetic) are simultaneously excited, the percentage of organic matter increases considerably, both in the dog and rabbit. This could not have been the result of the anæmia due to sympathetic excitation, since no such effect was observed by interrupting the blood supply of the gland.

As H. had previously shown, the microscopic appearance of gland cells is altered by a long-continued nerve excitation. This change is slight after excitation of the cerebral nerve, but more considerable and somewhat different after stimulation of the sympathetic, although the latter nerve, when stimulated alone, does not produce any parotid secretion whatever.

In conclusion, H. maintains the theory that there exist secretory nerves influencing the secretion of water and salts, and trophic fibres governing the secretion of organic matter. The former fibres predominate in the cerebral gland nerves; the latter are mostly contained in the sympathetic salivary nerves.

Some entirely new facts as regards the innervation of the submaxillary gland, have been found by J. N. Langley (*Journal of Phys.*, Vol. I., No. 1, p. 96), in the cat, an animal not yet examined in this respect. In the dog, as is well known, the saliva normally obtained by stimulation of the sympathetic nerve, is extremely viscid, containing much mucin; whilst that obtained by stimulation of the chorda tympani is more or less watery, containing a much less quantity of mucin. In the cat, however, the sympathetic saliva is less viscid than the chorda saliva. In dogs the chorda is paralyzed by a small quantity of atropia, but the sympathetic not at all (or only by excessive doses.—Langley). In the cat, on the other hand, the two nerves are almost alike in their susceptibility to the paralytic effects of atropia. In the dog, simultaneous stimulation of both nerves checks the secretion, the two nerves being apparently antagonistic to each other; but in the cat, minimal effective stimuli, when applied simultaneously to the chorda and sympathetic nerves, are not antagonistic, as regards secretion; on the contrary, the amount of secretion from the simultaneous stimulation is at least equal to the sum of the amounts from separate stimulation. The effect of stimulation of either nerve is even increased by faradizing the other nerve with a current by itself insufficient to provoke secretion. Only when both nerves are simultaneously exposed to *very strong* currents, the amount of secretion which the chorda alone can produce is somewhat lessened by the action of the sympathetic, probably from its influence on the vessels of the gland.

These researches were continued by Langley in a subsequent number (Vol. I., Nos. 4 and 5, p. 332) of the *Journal of Physiology* and extended to the antagonism of atropin and pilocarpin on the submaxillary gland of the cat. We can mention his principal results partly in his own words.

An exceedingly small quantity of pilocarpin, 0.001 gram., will call forth a lively secretion of saliva. A few minutes after the beginning of the secretion so produced, stimulation of the chorda causes an increase in the flow, which is much less than the flow caused by equal chorda stimulation previous to the pilocarpin injection. The effect of stimulating the sympathetic is in like manner diminished, but proportionately less.

After successive large pilocarpin doses there occurs a long continuing condition in which stimulation of the sympathetic causes no increase, but only a decrease of the secretion, more and more marked (in certain limits) with the increase in the strength of the stimuli and the time during which the stimuli are applied, and in which stimulation of the chorda causes a very slight increase in the secretion, rather more marked with the stronger stimuli. If a very large quantity of pilocarpin is brought into the gland either by direct injection into the gland (lingual) artery or into the gland duct, the secretion ceases at once, and the nerves are paralyzed for some time.

Atropin, in sufficient quantity, produces a complete stoppage of secretion and paralysis of the secretory nerves even after a previous dose of pilocarpin. The atropin effect can, however, be completely overcome by a subsequent injection of pilocarpin into the gland artery or duct. This mutual antagonism can be repeatedly shown, but is each time less perfect as far as the action of pilocarpin is concerned.

By stimulating either chorda or sympathetic (electrically) and causing a secretion, the atropin paralysis of these nerves, but especially of the sympathetic (which normally is less affected by atropin than the chorda), is more readily brought about. The atropin paralysis is also more readily effected after prolonged pilocarpin secretion, but after pilocarpin a somewhat larger dose of atropin is required to paralyze the chorda than is normally the case. There are two factors then to be considered in the paralysis: the amount of pilocarpin—this causes the atropin to produce its effects less readily,—and the amount of saliva secreted, which enables the atropin to produce its effects more readily.

The diminution of sympathetic effect produced by pilocarpin is unaffected by a small dose of atropin, unless some amount of secretion has taken place, but in the latter case the parts which the atropin attacks, be they nerve endings or gland cells, are more readily attacked by atropin, and an atropin paralysis adds itself to the pilocarpin diminution of sympathetic effect. But although pilocarpin in comparatively small quantity, when producing a secretion tends to paralyze the sympathetic, yet it but slightly affects it if a small amount of atropin (not enough by itself to paralyze that nerve) be given previously or at the same time.

As general conclusions, Langley adds:

Pilocarpin, in proportion to the quantity given, paralyzes both the chorda and sympathetic secretory fibres.

The diminution of the pilocarpin secretion caused by stimulating the sympathetic, is a direct effect of diminished blood supply, and not of nerve fibres inhibitory to the secretion.

The slight increase of saliva obtained by stimulating the chorda after a large dose of pilocarpin, is due, not to the action of its secretory, but of its vaso-dilator fibres. H. G.

NERVE DEGENERATION.—Notwithstanding the large number of researches on this subject, Dr. Th. Rumpf (*Unters. aus d. Phys. Institut d. Univ. Heidelberg*, II., 3, p. 307) undertook an investigation on the changes in divided nerves, as a sequel to his researches on normal nerves (this journal, Jan., 1879).

Twenty-four hours after division of the sciatic nerve of the frog, the peripheral stump shows an escape of myeline, involving the fibres usually up to the first annular constriction, though sometimes also further. The following interannular segment (or at least a part of it), is filled with granules and cannot be colored with osmic acid. The axis cylinder is not visible until after the removal of the myeline by means of alcohol and ether. It is then found considerably swollen, and terminating with a club-shaped end a short distance from the point of division. The swelling of the axis cylinder extends through one or more interannular segments. As Rumpf had shown previously, the axis cylinder, after separation from the centre, is tumefied and finally dissolved by lymph, hence the thickened club-shaped end. In this gradual swelling Rumpf seeks the motive force for the escape of myeline.

Forty-eight and seventy-two hours after the section the myeline has diminished in quantity, and is but feebly colored by osmic acid. The absorption of myeline is partly the work of migrating cells, which enter the nerve fibre and transport fragments of myeline. By this time more of the axis cylinder has also been absorbed, while the swelling extends further along its course. The same observations can be made on the central stump of the nerve.

The degenerative changes proper occurring in the peripheral trunk of the divided nerve are of long duration and slow course in the frog. Even seven to eight days after the division the axis cylinder is not yet absorbed wholly in the interannular segment involved in the section.

An entirely different course is observed if the nerve be divided at two places, *i. e.*, the portion observed be cut off from both centre and periphery.

While the metamorphoses of the myeline are as usual, the axis cylinder swells rapidly and is completely absorbed in the course of a few days after a double division of the nerve. Hence, Rumpf concludes that the axis cylinder by itself has not the ability to regulate its own nutrition, but that its nutritive regulation depends on its connection with the centre. After separation from the centre, a certain but insufficient nutritive influence is still exerted by the peripheral termination.

Ranvier has claimed that the loss of conductibility of a divided nerve is due to the interruption of continuity of the axis cylinder by the encroaching protoplasm (this journal, Jan., 1879). Rumpf, however, rejects this view, since a divided nerve loses its irritability in the frog within fourteen days, while no interruption of continuity of the axis cylinder can be demonstrated even after sixteen days. Hence, while the nerve is unable to conduct motor impulses, a nutritive impulse from the periphery can still traverse it.

The horn sheaths of the nerve persist for quite a time even after complete solution of the axis cylinder. Finally, however, they disintegrate into granules or plaques, and disappear.

In mammals degenerative changes pursue a faster course than in the frog. Still the axis cylinder is but little changed the second day after a simple division. Two days, however, after a double section, the axis cylinders are part swollen by the action of the lymph, and part completely dissolved.

THE COURSE OF SWEAT NERVES AND VASO-MOTOR FIBRES.—Induced by various contradictory statements, B. Luchsinger (*Pflüger's Arch.*, Vol. 18, XXXI., p. 483) has once more examined the course of the sweat nerves in the cat. He had previously claimed that the sweat nerves of the extremities were entirely derived from the sympathetic. This was confirmed by Ostroumoff and by Nawrocki. Adamkiewicz, however, and Vulpian had also found sweat nerves in the spinal roots of the extremity-nerves.

To decide this question a larger number of cats were examined. The abdominal sympathetic of one side was divided, and the animal either allowed to recover during some weeks or immediately tested, by placing it in a heated box. In the large majority the paw innervated by the divided sympathetic did not perspire in the least.

But in about one case in every six some sweat fibres did actually enter the nerves (of fore and hind extremity) through some route other than the sympathetic. In a second article, Luchsinger and Puelma (*Ibid*, p. 489) followed the course of the vaso-motor fibres of the sciatic nerve of the cat. As an unexceptional result they found that the nerves going to the vessels of the hind extremity are derived both from the sympathetic and the spinal roots.

DIRECT IRRITATION OF NERVE CENTRES has been studied by R. Marchand (*Pflüger's Archiv*, Vol. 18, XII., p. 511). In the first place, the frog's ventricle, separated from the greater part of the auricles, but still containing the ganglia situated at the auriculo-ventricular border, was compared with the ventricular muscle without ganglia. Mechanical, chemical, thermic or faradic irritation will induce but a single contraction in the heart muscle free from ganglionic cells, as it is obtained by isolating the lower part of the ventricle. If, however, the preparation still contains the ganglia, these stimuli cause a series of contractions. In the case of electric stimulation (a single induction shock) the number of pulsations increased directly with the degree of stimulation.

As a second test object, the spinal cord of decapitated frogs was employed. While a single induction shock applied to a motor nerve produces but a single muscle contraction, the same stimulus applied to the spinal cord provokes an irregular tetanus of some duration.

THE ACCELERATOR NERVES OF THE HEART.—Stricker and Wagner, *Mediz. Jahrb.*, Hft. 3, 1878 (abstr. in *Revue des Sci Méd.*).

It is now known that, in the dog, the acceleration of the pulse is produced by excitation of the *anse de Vieussens*, a nervous branch connecting the last cervical ganglion of the sympathetic to the first thoracic ganglion, or stellate ganglion. In order to find the real origin of the accelerator fibres of this branch, the authors isolated in the chest of the dog the trunk of the great sympathetic, by cutting its afferent branches and exciting it, commencing at the sixth thoracic ganglion. The acceleration thus obtained is due to the action of the current on the *anse de Vieussens*—since, if we ligate the trunk below it, only the upper segment remains excitable. As the acceleration is the more marked, the nearer the electrodes approach this *anse de Vieussens*, we may conclude that the accelerator fibres increase in number as we pass upward.

What is the origin of these accelerator fibres? Do they come from the ganglia themselves or from the cord, passing along the afferent branches of the ganglia? They should be referred to the cord. To demonstrate this, the authors divided the vagus nerve, and obtained an acceleration of the pulse which diminished after section of the two branches of Vieussens; the pulsations then became a little more frequent than at the beginning of the experiment. The acceleration following the section of the vagus is due to a tonus coming from the cord; and the existence of this accelerator tonus proves the medullary origin of the fibres which end in the anse of Vieussens.

The authors go still further: they obtained an acceleration of the pulse from excitation of the cervical cord; under the same conditions they obtained it also after division of the accelerator nerves, but more slowly and only when the blood pressure had been considerably raised. The acceleration due to excitation of the cord is therefore due to two causes — excitation of the accelerator nerves and increase of the blood pressure.

En resumé, the accelerator fibres arise in the cervical cord, following first a descending track, to ascend again in the form of *anses*, or loops, to the six upper thoracic ganglia, and end in the anse of Vieussens.

As to the functions of the accelerator nerves, numerous experiments, noted with care, have demonstrated to the authors that these nerves counterbalance normally the influence of the inhibitory nerves. These two orders of nerve fibres are antagonistic to each other, and a true interference of action exists between them. When they are cut the phenomena of interference become still more marked by the use of induction currents. The authors combat the assertion of Baxt, according to which excitation of the vagus renders the heart insensible to the influence of the accelerator nerves.

NERVE TERMINATIONS IN THE TONGUE.—Paul Lannegrace, *Thése d'agrégation*, Paris, 1878 (abstract in *Revue des Sciences Médicales*).

The study of the nerve terminations in the muscles of the tongue not having been undertaken by any histologist, M. Lannegrace devoted his personal researches in this direction. He found that the terminal organs of the motor nerves offered nothing very peculiar, but that the muscles of the tongue, above all the other muscles of the body, are rich in nerve fibres.

After passing rapidly over the terminations of the nerves of general sensibility, the author goes into details on the structure of the taste papillæ in man and other vertebrates. These organs are formed (1) of epithelial cells (gustatory cells), clearly differentiated from the epithelial covering of the tongue, by the softness and delicacy of their structure, and they present a striking analogy to the rods and cones of the retina or the olfactory mucous membrane; (2) of terminal nervous filaments, pale in color, and placed and located immediately under the epithelium. Continuity between the nervous element and the epithelium, has not yet been demonstrated.

Beginning the study of the distribution of the nerves in the tongue and their functions, M. Lannegrace, after having stated and discussed the older and more recent experiments relative to the physiology of these nerves, formulates the following conclusions:

Motor Nerves.—The hypoglossal is a motor nerve for the tongue, the anastomosis it receives from the first cervical pair furnishing it its motor fibres.

The glosso-pharyngeal controls the contraction of the pillars of the palatal vault.

The lingual possesses no motor fibres.

The facial presides to a very slight degree over the contraction of the stylo-glossus muscle.

The fibres of the chorda tympani pass (at least, in part) to the muscular fibres of the tongue, but act only in case of the deprivation of function of the hypoglossal.

Sensory Nerves.—The hypoglossal, ordinarily insensible at its origin, becomes sensory outside of the cranium; it possesses both a direct and a recurrent sensibility, due either to anastomoses with the vagus, the cervical plexus, or the lingual. Section of the hypoglossi does not, apparently, diminish the general or gustatory sensibility of the tongue.

The glosso-pharyngeal presides over the gustatory sensibility, especially for the perception of bitters in the posterior portion of the tongue. It is not the only gustatory nerve, but it is the nerve of taste *par excellence.* With the pneumogastric it supplies sensation to the base of the tongue.

The mixed lingual (which receives the chorda tympani) presides over the general sensibility and the sense of taste in the anterior two-thirds of the tongue, but takes only a small part in the perception of savors.

The chorda tympani possesses gustatory fibres, but it further has an indirect influence on the sense of taste, by virtue of the modifications it causes of the circulation in the anterior portion of the tongue.

M. Lannegrace closes his memoir by giving the various hypotheses of the origin of the chorda tympani.

ANATOMY OF THE DEPRESSOR NERVE.—This nerve, which was hitherto known only in the rabbit and the cat, was found by A. Kreidmann (*Arch. f. Anat. u. Phys.*, 1878, IV. & V., p. 405) also in man and other animals. In man the vagus consists of three or more bundles contained in one common sheath. From the most internal of these branches the superior laryngeal nerve arises. This nerve gives off a small twig, receives a second fine root from the vagus itself. So far, the arrangement is the same as in the rabbit. The difference, however, is that the depressor nerve, arising from these two roots, continues as a separate nerve in that animal, while in man it does not leave the sheath of the vagus, but returns to and re-enters the trunk of the vagus. The depressor fibres, however, are somewhat brighter in color than the rest of the nerve. In one cadaver out of six Kreidmann found the depressor nerve to continue for a distance of about 3 cm., as a separate nerve with distinct sheath.

A similar arrangement was found in the dog and sheep.

METALLOSCOPY ON FROGS.—In order to obtain a physiological basis for experiments on metalloscopy, Dr. H. Vierordt (*Centralbl.*, No. 1, 1879) studied the influence of metals externally applied on the sensibility of frogs.

The cerebrum was removed, to prevent spontaneous movements, and the animal fastened in an upright position. A round disc of zinc was applied to the abdomen (in one instance lead was used). The tests were begun fifteen minutes after removal of the hemispheres. The sensibility was tested by touching or compressing a toe of the animal and recording the number of times a reflex movement of that foot, or both feet, occurred. Each animal was tested during periods of twenty-five to forty minutes, alternately with or without metallic application. The figures given show a decided increase of reflex irritability by metallic applications. (The method, however, is very unreliable. A positive or negative result could easily be due to an (involuntary) inequality of mechanical irritation.)

The following papers have also recently appeared on the Anatomy and Physiology of the Nervous System:

HURD, The Physiology of Sleep, *Boston Med. and Surg. Journal*, Dec. 26, 1878.—IRELAND, Thoughts Without Words, *Journal of Mental Science*, Jan.—CARPENTER, Effects of Attention on the Bodily Organs, *Brit. Med. Jour.*, Dec. 14.—HOLL, on the Nervus Accessorius Willisii, *Archir f. Anat. u. Phys.*, Anat. Abtheil., 1878, VI. Hft.—KRONECKER and STIRLING, On the so-called Initial Contraction, *Ibid*, Phys. Abth., 1878, V. & VI. Hft.—VON KRIES, The Physiology of the Sense of Sight, *Ibid.*—BIRDSALL, The Embryogeny of the Sympathetic, *Archives of Medicine*, April.—ARLOING, Determination of the Excitable Points in the Cortex of the Hemispheres of Solipede Animals; Application to the Cerebral Topography, *Revue Mensuelle*, Mar. 10.—ADAMKIEWICZ, On the Physiology of the Sweat Secretion, *Virchow's Archiv*, LXXVIII.—OBERSTEINER, Experimental Researches on Attention, *Brain*, Jan.

b.—PATHOLOGY OF THE NERVOUS SYSTEM, AND PATHOLOGICAL ANATOMY.

THE DIAGNOSTIC VALUE OF THE TENDON-REFLEX.—Dr. A. McLane Hamilton, *Boston Medical and Surgical Journal*, Dec. 19, gives an account and analysis of eight cases of locomotor ataxia, in four of which the tendon-reflex was absent, as is stated to be the case in this disease by Westphal, while in the remainder it was present and in some cases even exaggerated. These latter were not less marked instances of the disease than the others, so far as the diagnosis could be made during life.

The deduction of Tschiriew that the absence of this symptom indicates a degeneration of the posterior columns at the horizon of the third and fourth roots of the crural plexus, and that when the morbid process does not reach as low as this, it may still be present, may perhaps account for the variations we observe, but further investigations and post-mortems are requisite before we can give this symptom its exact value in diagnosis.

DR. C. H. HUGHES, *St. Louis Medical and Surgical Journal*, Feb., also devotes a paper to this subject, in which he gives the result of the testing of this phenomenon in a number of healthy individuals, and patients suffering from nervous disorders, and concludes, that its absence is by no means necessarily diagnostic of any spinal disease, and only of importance when associated with other symptoms.

THE following is the abstract of a paper read before the Royal Medical and Chirurgical Society, Jan. 28 (rep. in *Brit. Med. Jour.*, Feb. 1), by Dr. W. R. Gowers, and entitled "A Study of the So-called Tendon-Reflex Phenomena," together with the discussion that succeeded it :

The paper recorded a series of observations on the two forms of muscular spasms commonly known under the above designation ; that normally produced in the quadriceps extensor of the knee on striking the patellar tendon (knee-reflex), usually absent in locomotor ataxy and excessive in lateral sclerosis of the spinal cord ; and that clonic movement which occurs at the ankle-joint on sudden dorsal flexion of the foot, in lateral sclerosis of the cord (ankle-clonus). To ascertain the characters of the knee-reflex, it was examined in three hundred patients, of whom one hundred and fifty were the subjects of simple epilepsy. Of the whole three hundred, it was absent on both sides in eighteen, of whom five had ataxy, three paraplegia without ataxy, one suffered from old hemiplegia, one from vertigo, one from tumor cerebri, one from old rickets, and the remaining six from epilepsy, five being girls. Two only of the eighteen were good walkers. Of twenty-seven hemiplegics, the reflex was equal on the two sides in thirteen, excessive on the weaker side in fourteen. In three cases, it was absent in one leg only ; two of these presented weakness of the leg in which it was present ; in the third it was absent in the leg in which the patella had been fractured and the two halves were separate. Of one hundred epileptics, with good walking power, the movement of the foot in ninety was between one and three inches; it was absent in none, excessive in six; the average was just two inches. Of twenty-two epileptics, with bad walking powers, it was absent in five and excessive in five. The reasons for regarding it as a spinal reflex were strong, especially its loss in spinal disease, its arrest by section of the crural nerve, its radiation to the other leg in animals, which the author had observed in several cases in man (and also a radiation to the back muscles); and, lastly, the graphic study of the movement. Tracings were shown, taken on a manograph-drum, the writing-pen being fixed to the foot. The effect of the tap and the muscular contraction were both marked, and were separated by an interval of .09 to .15 second, the common interval in health and disease being .10 or .11 second. This corresponded with the time necessary for a reflex action (conduction .045 second, latent stimulation .01 second, reflex process on spinal cord .05 second). Reasons were given for doubting the accuracy of the shorter interval obtained by Tschirjeff, with the foot fixed. The interval was sometimes occupied, in some cases the latter part only, by a slight rise, due to a muscular contraction, probably indicating a direct stimulation of the muscular fibres. As a spinal reflex, the question of the origin of the afferent impulse in the tendon, or in the muscle, by sudden tension, must be left open. Facts were mentioned pointing to the latter view. As a reflex, it was arrested by damage to the posterior nerve-roots (as in locomotor ataxy), by disease of the grey matter (as in muscular atrophy involving the quadriceps, sometimes, as in a case related, when slight only), or by damage to the anterior roots (as in old meningitis). It was also lost in advanced pseudo-hypertrophic paralysis. Its occasional persistence in locomotor ataxy was connected with slight damage to the posterior root-fibres, as was shown by sensation being little or not affected, and commonly by the absence of lightning pains (Buzzard, Westphal). A case of mixed lateral and posterior sclerosis was related, in which sensation was more affected, lightning pains greater, and knee-reflex lost in one leg; while in the other leg the knee-reflex was present, sensation was less affected, and lightning pains were slighter, though occurring. From forty tracings of the ankle-clonus, the regularity and time of the movement had been ascertained. From five to seven contractions occurred per second, the average being 6.1. During the intervals, the relaxation of the muscles was incomplete; a certain residual contraction persisting. A similar clonus might often be obtained on the plantar adductor pollicis, of which tracings were shown, having nearly the time of the ankle-clonus, and had been met with by the author in one case, and also a knee-clonus (in the quadriceps), having, however, a much slower time (2.5 per second). In the ankle-clonus, did the stimulus act on the tendon or on the muscle? It could not be excited by any stimulus applied to the tendon, which did not increase its tension, and, therefore, which did not act upon the muscle. A lateral tap on the tendon would not cause it, if the tendon were carefully supported on the other side. The tendon was very sensitive to a pinch; but this caused a quite different effect. On the other hand, the initial contraction could be excited by tapping the muscle, which could affect the tendon very little. The question of its reflex nature was next considered. The author had found that in cases in which the ankle-clonus could be obtained, and in slighter cases of similar central disease in which it was not obtainable, or had passed away, a tap on the anterior tibial muscles, during passive dorsal flexion, excited a contraction in the muscles of the calf, which did not occur when the adjacent tibia was similarly tapped, and so could not be the result of increased tension. This appeared at first to be of a true reflex nature; but, on taking tracings, the interval between the taps and the contraction was found to be only .03 to .04 second, insufficient for the mere conduction to and from the cord, and less than half that necessary for a spinal reflex. The stimulation of this muscle must be direct, by the vibration of the tap, which could indeed, even in health, be felt by the hand placed on the calf. Other measurements showed that, in lateral sclerosis, the interval between the tap on the Achilles tendon and on the muscle, and the resulting contraction (during passive dorsal flexion), was .025 to .04 second and .035 to .04 second respectively—quite insufficient for a reflex. Therefore, the stimulation of the muscle must be direct, not reflex; and, therefore, not from the tendon. But this did not exclude all reflex influence; the extreme irritability to local stimulation, excited by tension, might be reflex. Tension on muscular fibres caused an afferent impression (painful when long continued or after cramp), and tracings showed that the irritability was not developed instantly on the tension first being put on, but gradually, after a period long enough for a reflex. If the tension were previously put on, the first contraction was as strong or stronger than the rest. Hence it was understood why, though local, it occurred in spinal sclerosis. The reflex phenomenon in these cases on stimulating the skin or pinching the tendo Achillis was then considered (flexor tonic spasm exciting and broken by the ankle-clonus), and its relation to spinal epilepsy. The intervals between the contractions of the knee-clonus bore the same ratio to those of the ankle-clonus as did the interval between the tap and the isolated contraction of the knee-reflex, to the interval between the tap on the tendo Achillis and the contraction of the calf. Hence each contraction on the ankle-clonus was probably reflex, excited by the sudden tension on the muscle from the effect of the proceeding contraction. In the muscles of the calf and great toe the sequence of tension and contraction occurred at each step in walking, and a physiological reflex between the two was probably developed in the act of learning to walk. The author had never seen the ankle-clonus in spastic paraplegia in a child who had not walked. A reflex relation between tension and contraction probably played an important part in the co-ordination of often repeated movements, and its loss might be part of the pathological state in locomotor ataxy, in which the allied knee-reflex was also lost. The ataxic and the child learning to walk might thus be similar in this condition as well as in appearance. If this view of the ankle-clonus were correct, it should be a normal phenomenon excessive in disease. Four tracings of the movement, from four healthy individuals, were exhibited, showing the same uniformity and time as in disease. They were taken in the sitting posture, with the ball of the foot resting on the ground, when the ankle-clonus, set up voluntarily, would go on independently.

The President, in expressing the thanks of the Society to Dr. Gowers for his elaborate paper, suggested that it would be useful in such communications to denote the points which it was specially intended to illustrate or prove. Dr. Buzzard said that the paper dealt with two interesting phenomena, the "patellar tendon-reflex" and the "foot-clonus." As regarded the patellar reflex, the tendency of investigation was to show that its absence was an early sign of locomotor ataxy. The difficulty in standing with the eyes closed, pointed out as a test by Romberg, was of value; but, in its early stage of ataxy, this often was not present; and, in such cases, he believed the absence of patellar tendon-reflex would be very valuable. He had not yet found patellar tendon-reflex present in any typical case of locomotor ataxy; it had been retained in cases of ataxy or of ataxy combined with other symptoms; but was absent in ataxy accompanied with lightning pains. A very close connection would probably be found between the absence of patellar tendon-reflex and the characteristic pain of ataxy. As regarded the foot-clonus, if it were determined (as was very probable) to be an evidence of organic disease of the spinal cord, it would be an important means of distinguishing between trivial functional affections and grave structural diseases of the cord. He believed that Dr. Gowers' paper was the first in which the graphic method had been applied to the subject. He had been struck with the very probable explanation of ankle-clonus given by Dr. Gowers — that it was probably the pull on the intra-muscular nerves that caused the contraction. He was rather inclined to differ from Dr. Gowers when he said that the patellar tendon-reflex could be produced by a blow on the quadriceps extensor muscle. In some cases of ataxy, a blow on the vastus externus produced a slight upward movement of the foot, but this was different from the jerk produced by striking the tendon of a healthy man. He believed that the contraction produced when the muscle was struck, was due to the direct stimulation of the muscle itself. Dr. Andrew inquired as to the prognostic value of patellar tendon-reflex with lightning pains. He had met with a case in which the lightning pains had been present thirty-four or thirty-five years without any other symptoms of locomotor ataxy. The patellar tendon-reflex was absent; how long it had been so he, of course, could not say. Dr. Gowers said that the value of the absence of patellar tendon-reflex was diagnostic, not prognostic. Dr. Andrew's case was probably one of ataxy arrested in the first stage. Cases sometimes occurred in which the lightning pains persisted many years without other symptoms of locomotor ataxy. Almost all evidence was in favor of the connection between loss of patellar tendon-reflex and lightning pains. Absence of disorder of sensation, and absence of lightning pains indicated that the affection of the cord was slight. Perhaps, as an exception, a case might some day be met with in which the patellar tendon-reflex was preserved while lightning pains were present. A slight contraction might be produced in some cases by striking the muscle; but, when he spoke of exciting patellar tendon-reflex by a blow on the muscle, he referred to instances where the tendon-reflex was in excess, and where striking the muscle would cause a jerk of two or three inches in the same way as a blow on the tendon.

It will be perhaps remembered that in the issue of this journal for October, 1878, we mentioned a case in which all the symptoms of tabes had existed for years, the fulgurant pains included, and yet at the close of life the tendon-reflex was even exaggerated. In his recent lecture on the Diagnosis of Locomotor Ataxia (*Am. Clin. Lectures*, Vol. III., No. XII.) Dr. E. C. Seguin gives among the symptoms of the first stage of the disease, which designates the "stage of fulgurating pains," a *diminished* tendon-reflex at the knee, and includes its absolute loss as a symptom of the second or ataxic stage. It seems to us still that more light is needed on the subject before we can assign to this symptom its correct value in the diagnosis of the disorder. It has evidently been over-valued by Westphal in his first utterances in regard to it, and we now have an accumulation of more or less conflicting observations and opinions from various authorities.

A Prodromic Sign of Spinal or Cerebro-Spinal Affections.—At a recent clinic at La Pitie, M. Lasègue called attention to a point in a case of paralysis agitans which is worthy of note, and we therefore translate the paragraphs relating to it from the *Gazette des Hopitaux*, No. 4, in which they occur.

The subject of this observation is a man aged fifty-one, formerly a clerk, with no morbid antecedents, either personal or hereditary, and even of a robust constitution. A little more than eight years ago, in 1870, while he was at his desk, he was taken all at once, without any apparent cause, with a severe pain in the iliac region. This pain has lasted since then continuously, with frequent exacerbations.

Here M. Lasègue, interrupting the recital of the history of the case, called attention to the double peculiarity of the lack of morbid antecedents, and the outset of the disease, by a pain in the iliac region, near the anal fold, a local pain of an altogether peculiar character, not to be explained as a neuralgia, being on the track of no nerve and following the course of none, as known; not by any inflammation, nor by rheumatism, of which, moreover, it possesses none of the characters, being neither increased nor diminished by pressure, or modified in any manner whatever by any external agent. Whenever, said M. Lasègue, you meet in a man in such good condition as this one, without any known or appreciable cause, one of these *bizarre* pains, not explainable either by a neuralgia, an inflammation or rheumatism, which is not affected by pressure or by any external agency, you may say to yourself that you are in the presence of an incipient spinal or cerebro-spinal affection. This kind of pain is not specially characteristic of paralysis agitans; it is a prodrome common to spinal diseases, as we see in the fulgurant pains of locomotor ataxia and the pains that precede general paralysis. Its explosive appearance does not, therefore, suffice to indicate the character and nature of the disorder of which it is the first manifestation; but it certainly announces the imminence of a cerebro spinal affection; it is, to adopt M. Lasègue's picturesque expression, only the telegraphic signal announcing the message, of the contents of which it gives no indication.

The Differential Diagnosis of Neurasthenia.—The following is from the abstract of a paper by Dr. G. M. Beard on Neurasthenia, read before the New York Neurological Society, January 6, as given in the *N. Y. Med. Record:*

Dr. Beard regarded it important to make a differential diagnosis between neurasthenia and organic or structural disease of the brain or spinal cord. He had been frequently consulted by physicians, with reference to themselves, for symptoms which were supposed to indicate ataxia or some form of organic disease of the spine or brain, when in reality they only had symptoms of neurasthenia. Some of those medical men were greatly alarmed, and the more they read upon the subject, in German authorities, the more alarmed they were: for, in our literature, the distinction between neurasthenia and symptoms of organic trouble were not clearly made out.

Four points of differential diagnosis between neurasthenia and organic disease of the brain and spinal cord.—There were four points in the differential diagnosis of neurasthenia from organic disease:

1. The symptoms of organic disease were usually fixed and stable. Those of neurasthenia and allied states were fleeting, transient, fluctuating, metastatic, recurrent, intermittent.

2. There were certain symptoms of neurasthenia which did not usually appear in organic or structural disease. Of those mention was made of general or local itching without apparent cutaneous disease; tenderness of the teeth and gums; special idiosyncrasies with regard to food and medicine, which did not exist prior to the illness; morbid desire for stimulants and narcotics; morbid fear in its different phases; agoraphobia (fear of places); astraphobia (fear of lightning); anthrophobia (fear of men); likewise sick headache.

3. In organic disease reflex activity was usually diminished, while in functional disease reflex activity was usually increased. To that rule there were some exceptions, as in spasmodic spinal paralysis.

4. Neurasthenia and allied troubles were most likely to occur in those in whom the nervous diathesis predominated.

The characteristics of the nervous diathesis he had frequently described in other writings.

Neurasthenia was to be distinguished from hypochondriasis or pathophobia, from hysteria, and from cerebral and spinal anæmia and hyperæmia.

In neurasthenia the anæmia and hyperæmia of the brain and spinal cord were results; symptoms, temporary, intermittent, and not the disease; and it was unphilosophical to call them the disease.

The same was true with reference to oxaluria, a condition often found in neurasthenia.

The doctrine that innervation preceded circulation was a growing one among neurologists.

That point was urged in his original paper on neurasthenia. Vulpian's researches upon the physiology of sleep were in harmony with that view. Erb, in his chapter on neurasthenia, also leaned towards that view.

Neurasthenia was also to be distinguished from nervous syphilis, which it sometimes simulated in a wonderful way.

Neurasthenia also occasionally simulated in a perfect and interesting way the *symptoms of a common cold.* The chilliness, the positive coldness, the tremor, the heaviness, the soreness, the pain in the back and the limbs, and, in some cases, the excessive secretion from the eyes and nostrils — all made it hard to determine whether the patient had taken cold or not.

Over-dose of electricity. —An over-dose of electricity upon a neurasthenic patient might bring on all the symptoms of a common cold, so that the patient might suppose he had taken cold.

Neurasthenia might simulate rheumatism.

Differential diagnosis between neurasthenia and anæmia.—Neurasthenia was to be distinguished from anæmia mainly by the following symptoms:

Neurasthenia was found chiefly in connection with the nervous diathesis; the patient might be plethoric; the pulse might be full and normal; no cardiac murmurs; no pallor; was common to both sexes, but not so relatively frequent in females; was benefited by remedies which directly affected the nervous system, iron alone being of but little service; recovery was gradual, and occurred chiefly between the ages of fifty and sixty.

Anæmia, on the other hand, appeared always with the tuberculous, the rheumatic or other diathesis; was marked by increase of the watery constituents of the blood and diminution of red blood corpuscles; was found in all periods of life, from infancy to old age; was accompanied by small, weak pulse, cardiac murmurs, pallor of the face and lips, less mental depression than was present in neurasthenia; occurred relatively more frequently in females than in males; might be rapidly cured by removal of the cause, and was benefited by iron alone.

The two conditions, anæmia and neurasthenia, were sometimes associated in the same patient.

Differential diagnosis between neurasthenia and hysteria. — These two diseases were oftentimes confounded with each other. In hysteria there were usually convulsions, paroxysms, *globus hystericus*, anæsthæsia of the epiglottis, ovarian tenderness, attack of general and local anæsthesia, a certain acuteness, intensity and violence of the symptoms; was usually associated with great emotional activity; occurred most frequently in non-balanced mental organizations, and was comparatively rare in males. In the mental or psychical form it occurred with perfect physical health. Recovery might be sudden under purely emotional treatment.

In neurasthenia, on the other hand, there were no convulsions or paroxysms, no *globus hystericus*, no anæsthesia of the epiglottis; there might be ovarian tenderness — often was — but it was not so common as in hysteria; the attacks of local or general anæsthesia were not so common; the symptoms were more moderate, quiet, subdued, passive; often occurred in a well-balanced intellectual organization; very common in males; always associated with physical debility; recovery was never sudden, but always gradual.

Dr. Beard agreed with Erb that the disease needed a more systematic and careful study than it had received. It was very common, was increasing in frequency, and was the cause of a great deal of real distress. He then read the histories of a number of cases which illustrated the symptoms mentioned. In some of these the diagnosis of ataxia had been made.

The society then adjourned.

Diagnosis of Disease of the Spinal Cord.—In the Society of Physicists and Physicians, Dresden session, February 9, this year, privy councillor Dr. Erdmann delivered a lecture on the diagnosis of disease of the spinal cord.

After some introductory remarks regarding the progress of anatomical knowledge of the diseases of the cord and its congruousness with clinical observations, the author proceeds to consider some accurately characterized groups of symptoms, from which one may diagnosticate definite diseases of the cord as to the *gait ;* the author distinguishes the *paralytic* gait, which, dragging along, like that of great weariness, belongs to certain forms of chronic myelitis; further, the *atactic* gait with hesitating movements, and the well known uncertainty of patients in turning about, and, finally, the *spastic* gait, caused by reflected muscular tension.

Among other symptoms Erdmann mentions the changed condition of the *tendon-reflex*, which is increased in lateral sclerosis, and diminished in tabes, where it is at times entirely absent; further, the *trembling* during intentional movements — the so-called intention trembling; then the *disturbances of voice and deglutition*, as especially characteristic of bulbar paralysis, the atrophies of the optic nerve, diplopia, formication, especially in the ulnar region; the pain in the heel and the gastric crises to which Charcot attaches so much importance.

Complicated cases and certain transition forms produce also complicated groups of symptoms, whose significance is not so easy to determine and to explain, in which we must still have refuge in a spinal irritation, which, though not anatomically founded, nevertheless certainly does exist.

Besides *spinal pain*, which always presents as the first symptom, all other possible symptoms may occur, as in organic diseases of the spinal cord. Congestions cause pains in the region of the spinal column, which, however, do not increase with pressure upon the spinous processes; and, further, paræsthesia, anæsthesia, conditions of depression, irritation of the sexual organs, etc. — all these symptoms are aggravated in the dorsal, and diminished in the abdominal posture.

Anæmia of the spinal cord, as experimentally produced by ligation of the abdominal aorta, and as observed clinically, in consequence of embolic occlusions, is better known in its manifestations.

Leyden distinguishes three forms of spinal irritation, namely: 1—the hysteric; 2—the hypochondriac, and 3—the anæmic, to which are attached certain sub-varieties. The author observes that the difference between hysteria and spinal irritation is, in general, difficult to distinguish, and the beginning stages of disseminated sclerosis are also difficult to differentiate from hysteria. But in hysteria there is more disturbance of sensation, while in sclerosis there is more of motion. Among these motor disturbances the so-called *intention trembling* is especially to be remarked. It disappears during rest, which fact distinguishes it from paralysis agitans.

This tremor in intended movements lessens as the disease advances, and finally gives place to peculiar spasmodic contractions, especially of the extensor muscles, by which marked abnormalities of the joints are occasioned.

In hypochondriacal paralysis the subjective troubles are much more marked than those which are demonstrable as objective. Moreover, the paralyses are only slight; they change about and rapidly disappear. Aggravations are occasionally induced by dyspeptic disturbances. The basis of these difficulties is a venous hyperæmia of the lower part of the cord.

Patients suffering from the anæmic forms of spinal irritation are for the most part pessimists. True, paralyses are for the most part absent. There is only functional weakness, often caused by previous losses of semen to great extent. Pains occur in changing places; the bladder and rectum remain unaffected.

The general condition in real diseases of the cord remains often enough perfectly good for years, while the opposite is the case in spinal irritation. —*Deutsche Zeitschrift f. prakt. Medizin*, Nov. 23, 1878 (*Lancet and Clinic*).

LOCOMOTOR ATAXIA.—Dr. A. Takacs publishes in the *Centralbl.* (50, 1878) the following clinical and anatomical results of his investigations on locomotor ataxia, promising a speedy publication of the details and methods.

1. The grey degeneration of the posterior columns, existing almost always in tabes, is a secondary process. The primary affection is an atrophy of the posterior roots, or the posterior cornua, or a posterior meningitis.

2. The constant anomaly of sensibility in tabes is a diminished velocity of conduction. Anæsthesia, however, or hyperæsthesia, do not occur in all cases.

3. The posterior columns contain centripetal fibres, nerves of touch. The grey substance conducts in the normal state only painful impressions (Schiff), but in morbid changes of the posterior columns it can assume the function of the posterior columns (Friedreich). This accounts for the diminished conductibility.

4. In a normal movement the muscles do not contract suddenly, but in variable phases, according to the intended movement. For this purpose the muscles require a graded stimulation, originating in the sensitive nerves, which perceive the muscle contraction.

5. But if this stimulation, starting in the sensory nerves, is retarded, the muscular contraction becomes also retarded, and hence jerking—*ataxic.*

6. In tabes the retardation of sensitive conduction is due to the fact, that the slowly conducting grey substance is fulfilling the function of the degenerated posterior columns.

7. When the posterior columns alone are degenerated, only ataxia occurs; but if the degeneration involves also the posterior roots and cornua, there will be found anæsthesia, or hyperæsthesia, according to the stage of the disease.

CONTRACTURES IN INTRA-VENTRICULAR EFFUSIONS.—At the session of the Societé de Biologie, March 1 (rep. in *Le Progrès Médical*), M. Cossy related some experiments in which he had injected liquids directly into the ventricles of the brain. M. Duret had accounted for the contractures observed in intra-ventricular hemorrhages, and in experimental injections of the ventricles, by presuming an excitation acting on the parts forming the floor of the fourth ventricle, especially the restiform bodies. M. Cossy rejected this explanation, since in his experiments he had found the coagulated liquid injected only in the lateral and middle ventricles, and yet the contracture had been produced. He therefore concluded that it must be due to the irritation of the subjacent excito-motor parts (internal capsule and cerebral peduncles) by the sudden compression of the walls of the lateral and middle ventricles.

In like manner he explains the contracture in cases of hemorrhage limited to the lateral ventricles; not admitting the explanation of Duret that it is caused by the shock transmitted through the cephalo-rachidian fluid to the floor of the fourth ventricle. He remarked that convulsions were not any more frequent in cases where the effusion had reached the fourth than in those in which it remained limited to the lateral ventricles. Out of twenty cases of ventricular hemorrhage in which the blood reached the fourth ventricle, in eight only were there convulsions, in twelve they were lacking.

HYDROPHOBIA.—T. B. Curtis, *Boston Medical and Surgical Journal*, Nov. 7 and 14, 1878, gives an account of a case of hydrophobia very well observed, and follows it with a discussion of the pathology of the disease. After passing in review the various causes of the paroxysms and their *facies*, he expresses the opinion that there are no involuntary convulsions whatever in hydrophobia, but that the spasms "consist in sudden attacks of *paralytic apnœa*. due to temporary, partial or complete, inhibition of the respiratory centre, taking place under the influence of peripheral impressions. The inhibitory stimulus proceeds first from the area of distribution of the superior laryngeal nerve, being originated by attempts to drink and by accumulated saliva; secondly, from the area of the fifth pair, as a result of wetting the lips or face, and of fanning; thirdly, from the nerves of sensation of the trunk and limbs; fourthly, from the nerves of special sense; and fifthly, from the emotional regions of the hemispheres; a paroxysm being excited by the fear of a paroxysm." Thus the characteristic phenomena of hydrophobia consist in merely the exaggeration of a normal physiological process —that of respiratory arrest or inhibition.

That it is a diminished resistance of the respiratory centre, and not an increased stimulus that is at the bottom of these effects, he thinks is indicated by the general permanent diminution of respiratory activity observed in these cases of hydrophobia, which is due to actual structural changes in the medulla. This is aided in its morbid action by additional depressing influence from the emotional cortical regions.

The following is therefore his scheme of the pathology of the disease:

"The activity of the respiratory centre—being kept in a permanent state of depression, 'below par,' so to speak, in consequence of local alterations of structure, and also in consequence of depressing influences proceeding from the diseased emotional regions of the brain—is thereby rendered liable at any moment to become reduced to zero (paroxysm of paralytic apnœa) by slight inhibitory stimuli of peripheral origin; death eventually resulting, during or after a final paroxysm, from entire failure of the inspiratory forces."

Dr. J. J. Putnam, in the number of the same journal immediately succeeding those containing Dr. Curtis' paper, discusses the same subject, and disagrees with the former author in that he considers that diminished resistance power of the respiratory centre to inhibitory influences as of less weight, and increased inhibitory stimulus as of more weight in the production of the paroxysms. A great deal of influence is ascribed by him to emotional influence, and he considers that it is probable that the case steadily progresses to a fatal termination in the intervals between the paroxysms, and that the attacks only hasten, not cause the fatal result.

Dr. W. R. Gowers, *Brit. Med. and Surg. Journal*, Feb. 6, thinks that the paroxysm is due to increased stimulation of that part of the respiratory centre that has to do with extraordinary respiratory efforts, an over action of this centre on reflex excitation, the over action being of the nature of diminished resistance not to inhibitory, but to excitatory stimuli. He considers the order of the action of the hydrophobic poison is about as follows: first, on the medulla oblongata; second, on the cerebral hemispheres; third, on the spinal cord. The effect on the latter is rarely marked except in the later stages of the disorder; the effect on the hemispheres is shown in the delirium that is so noticeable in some cases; while the action on the medulla is prominent throughout.

TETANUS.—The following are the conclusions derived from an analysis of four hundred and fifteen cases of tetanus, by Dr. D. W. Yandell, *Brain*, No. III., Oct., 1878.

I. That traumatic tetanus is most fatal during the first decade of life.

II. That it usually supervenes between four and nine days after the injury.

III. That the largest number of recoveries are found in cases in which the disease occurred after the lapse of nine days from the injury.

IV. When tetanus continues fourteen days, recovery is the rule and death the exception, *apparently independent of the treatment.*

V. Tetanus arising during the puerperal state is the most fatal form of the disease.

VI. Chloroform has up to this time yielded the largest percentage of cures in acute tetanus.

VII. The true test of a remedy for tetanus is its influence on the history of the disease: (*a.*) Does it cure cases in which the disease occurred prior to the ninth day after the injury? (*b.*) Does it fail in cases whose duration exceeds fourteen days?

VIII. Tried by these tests, no agent has yet established its claim as a true remedy for tetanus.

NOCTURNAL EPILEPSY.—The following are the closing paragraphs of an interesting paper, by M. G. Echeverria, in the *Journal of Mental Science*, Jan., in which is given a pretty full discussion and description of the forms of nocturnal epilepsy. The article is of interest both in a clinical and medico-legal point of view, but its character and length are such that we cannot well reproduce more than its general conclusions.

"Nocturnal epileptic fits are observed more frequently in females than in males; and appear almost always associated with diurnal vertigo, *petit mal* or *haut mal*, when not with epileptic insanity.

"The ætiology of nocturnal epilepsy is essentially encephalic, and it may be chiefly attributable to hereditary predisposition, traumatisms of the head, alcoholism, syphilis, and strong emotional causes.

"The nocturnal incontinence of urine, the lacerations of the tongue, and the petechial eruption over the face and neck, are not constant phenomena, but, when existing, they possess an unquestionable pathognomonic value.

"The sudden explosion of frantic momentary bewilderment in the middle of the night, during sleep, or of insanity on arising in the morning, are proofs of nocturnal epilepsy. If nocturnal incontinence of urine, hereditary predisposition, with strange peculiarities of character, extreme propensity to anger and furious violence, are observed besides, these phenomena then prove, beyond all doubt, the existence of epilepsy.

"Most sleep-walkers and somnambulists are persons of neurotic temperament, exhibiting manifest signs of some neurosis, and ultimately arriving at unmistakable epilepsy or insanity.

"The attacks of somnambulism seldom, if ever, present the short duration, or the final outburst of violence characteristic of the nocturnal epileptic fits with talking and moving about of the patient. Nor do the former show the uniformity and constant sameness of the latter.

"Hallucinations chiefly of sight and hearing, of a most frightful, terrifying character, usually accompany and induce the wild excitement of the nocturnal fits.

"The nocturnal epileptic shows, as a rule, complete amnesia of his doings during the attack, but keeps more or less vivid recollections of his delusions, as of a dreadful nightmare.

"The nocturnal epileptic, acting like the somnambulist, in an unconscious automatic manner, cannot be held responsible for any misdeed perpetrated during his fits; he, however, must be regarded, in such cases, as one of the most dangerous lunatics, and restrained in a lunatic asylum.

"Nocturnal fits accompanied by paralysis, are free from immediate concomitant insanity.

"Finally, diligent and close inquiry into the phenomena of nocturnal epilepsy confirms the correctness of Trousseau's aphorism '*Tout ce qui est accident nocturne doit faire songer à l'epilepsie.*'"

Incontinence of Urine in Children.—At the meeting of the Harveian Society of London, November 20th, 1878, Dr. Farquharson read a paper on incontinence of urine in children, of which the following abstract is given in the *British Med. Journal*, December 28th:

After some preliminary remarks on the bearings of incontinence of urine on surgery and obstetric medicine, he referred to the subject under three headings. In some cases, this affection is found in children of pale, weakly organization, depressed and languid, and feeling keenly their infirmity. Here there is, no doubt, some weakened condition of the sphincter vesicæ, or of the nervous centres in the lumbar cord; and tonic remedies, and more especially small doses of wine, will usually act with excellent effect. Secondly.—There were cases of much greater severity, usually dating from soon after birth ; and here it is necessary to make a distinction between the enuresis by day and that by night, for the latter is much more difficult of cure than the former, and frequently resists all medicinal treatment—departing, if it do so at all, spontaneously about the period of puberty. The remedies which have been generally spoken of as most deserving of confidence, are those which act on unstriped muscular tissue, and of these, belladonna is the only one which, in the experience of the author, has given good results. It is necessary to give full doses ; and two ounces have been administered to a boy of seven before success—and even then only temporary success—was obtained. Ergot has proved disappointing, and santonin has been entirely without influence under the morbid condition. Class three includes those cases which may support the belief that incontinence of urine is truly a neurosis; for here we find this symptom coinciding with, and even alternating with other nervous lesions. Thus, on two occasions, it was observed concurrently with eczema, and once a very long-standing case was attacked with chorea, during the continuance of which perfect control over the bladder was regained. Nervine tonics are of little use here ; but the careful use of galvanism seems specially indicated, as well as blistering over the fifth lumbar vertebra, where modern experiment (ably summarized by Farr in the *Practitioner*) has shown the motor centre to be situated. The recently proposed plan of excluding meat from the dietary, was not found to be of much service, no special acidity of urine being ever observed to require the counteracting agency of purely non-nitrogenous food.

THE PATHOLOGY OF TETANUS.—G. A. Woods, *London Lancet* (Am. Rep.), January, gives the *post mortem* examination of the cord of a patient who died of traumatic tetanus, with comparisons with the appearances observed by others in this disease. The appearances in his observation were chiefly capillary dilatation and traces of granular degeneration in the posterior columns. The pathology of tetanus, according to him, is an increased reflex excitability of the spinal centres, arising from various causes, and producing finally structural changes. He says, in concluding : To sum up, therefore, I consider tetanus to be a disease which may be produced not by one, but by several causes, which, when once set up, lead to the following changes:

a. Enormous dilatation of blood-vessels in the medulla, particularly in the neighborhood of the hypoglossal and pneumogastric nuclei, and in the spinal cord, especially around the central canal and the portion nearest the seat of injury, the part in which colloid bodies are usually noticeable.

b. Leucocytal infiltration in the same regions; but according to Dr.

Gowers, who has examined my microscopical slides, much more uniformly distributed than in hydrophobia.

c. Granular and subsequent changes, the result of mal-nutrition, and most noticeable in the posterior columns, and varying according to disease duration.

N. B.—These microscopical deductions are from an examination of four cases.

That this condition is:

a. Associated with an exalted action of the ganglionic cells, originating in a point of the cord nearest the seat of injury, and extending upwards towards the medulla, the central seat of bilateral reflex action.

b. That the blood destined to supply the rapid ganglionic metamorphosis in the medulla, is either altered in quantity or quality, or in both.

c. The ganglionic cells of the medulla, not receiving their blood-nutriment in proper quantity or quality, can no longer supply that nutritive force which is so necessary for the proper and healthy performance and maintenance of its vital functions.

A NEW SYMPTOM OF IRRITATION OF THE FACIAL NERVE.—Dr. Leube describes (*Aerzt. Intellig.-Blatt*, No. 53) the case of a woman, aged sixty-two, who had been suffering from spasmodic tic doloureux for two or three months. It began with conjunctivitis, which was followed by blepharospasm; the spasm then spread over other branches of the facial nerve. The platysma myoid was the first muscle affected by the spasm, then followed the other muscles of the face. The spasms were so violent that the patient declared she felt as if her whole lower jaw were being torn away. At first, there was profuse salivation, but this gradually decreased. The spasms were clonic bilateral, and extended to all the muscles supplied by the facial nerves, specially to the orbicularis palpebrarum and the platysma myoides. The paroxysms followed in quick succession, but sometimes ceased altogether for several hours. When a paroxysm reached its climax, the patient sometimes uttered a peculiar sibilant sound, which could be produced by drawing the soft palate upwards, at the same time contracting the uvula and performing expiration. It was, therefore, at once suspected that, even while the spasm lasted, the muscles of the palate were convulsively contracted. This supposition was verified by subsequent laryngoscopic examination during the spasm. If the tongue were slightly depressed, it was easily seen that, when the paroxysm was very violent, the palate, which until then had been apparently in a rather relaxed condition, suddenly was contracted and drawn upwards, and the uvula, contracting also, almost disappeared. Dr. Leube, therefore, supposes that the sibilant sound, as well as the spasmodic contraction of the palate, were due to the part which those branches of the facial nerve that supply the palate took in the spasm. It was not possible to prove whether those fibres of the facial nerve which stimulate the secretion of the saliva, were also excited. The treatment consisted in giving Fowler's arsenical solution, either internally or hypodermically, and the results were most gratifying.—*British Med. Jour.*, February 1, 1879.

EPILEPSY AND EPILEPTIFORM SEIZURES. — Dr. J. Hughlings Jackson, in a recent paper on the Diagnosis of Epilepsy, *British Med. Journal*, January 11, gives as follows the principal clinical distinctions between epileptiform seizures and epilepsy:

Partly in order to show that I do not advocate the absolute separation of clinical from scientific investigation, but only the temporary separation, I will consider several broad differences and resemblances betwixt the two divisions of cases from a scientific as well as an empirical stand-point.

a. As to affection of consciousness.—The distinction is not that consciousness is lost in one and not lost in the other, but that consciousness is lost at first, or very soon, in the paroxysm of epilepsy proper, and late, or not at all, in the epileptiform seizures.

To say that an epileptic paroxysm begins with loss of consciousness, is equivalent to saying that the epileptic discharge begins in some part of the highest of the cerebral centres. This is an irresistible inference, be those highest centres where they may.

b. As to spasm or convulsion.—Convulsion begins more nearly bilaterally in the former; is more nearly universal at once, is more rapid, and is soon over. In the latter, it begins very locally on one side—hand, side of face, or foot—and becomes universal more gradually, the spasm being comparatively slow, especially at the outset.

c. As to seat of changes.—The latter depends on disease in some part of Hitzig and Ferrier's region; the former—this is hypothetical—on disease in some part in front of that region, or behind Ferrier's sensory region; that is, on disease in some part of what I suggest are the only, I think, middle motor and middle sensory cerebral centres; the parts in front of the middle motor centres being the highest motor centres; those behind the middle sensory centres, the highest sensory centres.

The vague term, "disease," or "changes," has to be analyzed. The term, "disease," is frequently used without regard to the distinction betwixt the two things, physiological abnormality (*d*), and abnormality of nutrition (pathology) (*e*).

d. As to abnormal physiological condition of centres.—In each, of necessity, there is increased instability of cells, since there occasionally occur excessive liberations of energy; in each there is a "discharging lesion." There is, then, no essential difference in the abnormal physiological condition in cases in the two divisions; yet the paroxysms of epilepsy proper are more "intense" (more spasm in a short time) than those of epileptiform seizures; and this possibly depends in part on the fact that the highest centres have most small cells. (Compare *b.*)

e. As to pathology.—In each there is increased nutrition; the excessive liberation of energy in the paroxysm of necessity implies increased taking in of nutrient substances having potential energy; increased nutrition is the other side of high instability. We have, as yet, little more than this very general, but inevitable, inference as to pathology in either. How the increased nutrition is brought about, we do not know in most cases; in some cases (see *f*), it is determined by tumor.

f. As to morbid anatomy.—We can say that the former is rarely caused by gross organic disease; the latter are often so caused, syphilitic gross disease in particular. Sometimes, however, in cerebral tumor there are attacks difficult to distinguish from the epileptic vertigo of epilepsy proper.

Of course, the gross organic disease only "causes" the seizures in the sense that it leads to high instability of cells in its neighborhood; it acts like a foreign body. The probability is that it leads to the hyper-physiological condition of instability (see *d*), by producing a sub-inflammatory change, and thus increased nutrition (see *e*). This difference as to morbid anatomy in the two seizures, can only mean, of course, either that gross organic disease most often occurs in some particular places, or that in some particular places it more easily leads to instability of nerve cells.

Several of the foregoing are, to a great extent, but detached statements from different stand-points of what Bright and Wilks long ago asserted, viz., that epileptic or epileptiform attacks, without loss of consciousness, point to gross organic disease, such as tumor of the brain.

As implied, however (*a* and *c*), the essential difference betwixt the two kinds of seizures is chiefly as to the degree of evolution of the centre, part of which has become unstable.

This leads me on to say that several of the above statements (*a*, *c*, *d*) as to difference in the two seizures, are implied in the following highly interesting generalization by Herpin: "*En résumé: plus le début est long moins la crise est violenté; plus, il est instantiné, plus, l'accés est intense.*"

ACUTE LUNG DISORDERS FOLLOWING HEMIPLEGIA. — The following abstract by Erlenmeyer of a memoir by Rosenbach, of Breslau (*Berliner klin. Wochenschrift*, 1878, No. 41), is taken from the *Centralblatt f. Nervenheilkunde*, 1878, No. 12:

"The author deems himself justified, on the basis of a relatively large series of observations—eleven cases—in concluding that acute lung affections, occurring just after unilateral paralysis from cerebral hemorrhage, *always occupy the paralyzed side.* He alludes to the well-known experiments of Brown-Sequard and Schiff, in which, after certain definite injuries to the brain, hyperæmia and hemorrhages of the lung of the opposite side were observed; and he furthermore mentions briefly—too briefly, indeed—the confirmatory fact that in many cases of hemiplegia the respiratory muscles of the paralyzed side are either weakened or altogether involved, and put out of action (Hasse, for example, in his text-book, II Ed., page 426, speaks of cases in which the respiratory movements of the paralyzed side are carried on altogether and alone by the diaphragm. The memoir of Berger on the paralysis of the long thoracic nerve, may be quoted here; he found paralysis of the levator anguli scapuli and trapezius on the paralyzed side of a hemiplegic patient *Ref.*) and seeks the explanation of the phenomenon in question in a *diminished reflex irritability* of the air-passages of the paralyzed side, which he considers analogous to the unilateral reflex inhibition of the external auditory passage, the nasal mucous membranes, the cornea, the cremaster, the abdominal superficies, and the musculature of the nipple. The supposition of such a reflex inhibition in the air passages is certainly allowable, and draws considerable support from the mechanism of the so-called *Verschluckungspneumonia*. Whether this condition is alone sufficient for the explanation of the processes in question, and whether the other causes deserve so summary a dismissal, seem to me still open questions. If once we allow the conclusion from analogy that diminished reflex of the air passages may cause paralyses of the bronchial and vascular muscular apparatus, and consequently venous hyperæmia and retarded circulation on the paralyzed side, then, and this applies especially to old cases, we cannot leave entirely out of consideration the facts of pathology which teach us that a destructive and function-inhibiting influence of a diseased cerebral hemisphere acts upon the lung of the opposite side. Nevertheless, the memoir of Rosenbach is a very meritorious one."

In addition to the above criticism of Erlenmeyer, we would add for ourselves that, apart from *a priori* considerations, which he does not seem to take much into account, eleven cases furnish but a very narrow basis for a generalization, such as that in which the author indulges. Moreover, there are in medical literature some contradictory statements, such as that of Hughlings Jackson, that certain pulmonary congestions and hemorrhages, occurring with cerebral hemorrhage, occur, with rare exceptions, in both lungs; and if one lung is more affected than the other, it is generally the right. (*Reynolds' System of Med.*, II., p. 526.) Though this assertion may be incorrect, yet considering the high authority of Dr. Jackson, a positive statement that the lung disorder *always* occurs on the paralyzed side, based on only eleven observations, appears like a rather incautious generalization.

There are other points in regard to which Rosenbach's views appear to us open to criticism, but as we have not at hand his original paper, we pass them by. His memoir is certainly not conclusive as to anything.

The following are the titles of additional papers on the Pathology of the Nervous System and Mind, and Pathological Anatomy:

Messemer, A Contribution to the Diagnosis of Neoplasms of the Pons Varolii and the Medulla Oblongata, *Physician and Pharmacist*, Jan., 1879. —Myers, The Influence of the Nervous System on the Health of the Mouth, *Lancet and Clinic*, Dec. 21.—McAldowie, On Spinal Epilepsy, *Brit. Med. Jour.*, Dec. 21.—Wilks, Remarks on Hemianæsthesia and its Cure, *Ibid*, Jan. 18.—Beard, Nervous Diseases connected with the Male Genital Function, *N. Y. Med. Rec.*, Jan. 25. — Hamilton, The Hysterical Simulation of Organic Nervous Diseases, *St. Louis Med. Journal*, Jan.— Guiteras, Two Cases of Anæsthesia, with some Metalloscopic Experiments, *Phil. Med. Times*, Feb. 15.—Caddy, The Identities of Cerebro-Spinal Meningitis, Dengue and Yellow Fever, *London Lancet* (Am. Repr.), Feb.— Beach, The Diagnosis and Treatment of Idiocy, with Remarks on Prognosis, *Ibid.*—Beard, The Nature and Diagnosis of Neurasthenia (Nervous Exhaustion), *N. Y. Med. Journal*, March, 1879.—Mills, Notes on the Localization of Diseases of the Brain, *Phil. Med. Times*, March 1.—Charcot, The Diagnosis of the Imperfect Forms of Multiple Sclerosis, *Progrès Med.*, Feb. 8.—Snell, Oligoria (Defective Sensibility in the Insane), *Allg. Zeitschr. f. Psychiatrie*, LIII., VI.—Witkowski, Remarks on the Chorea of the Middle Ages, and on Psychic Infection, *Ibid.*

c.—THERAPEUTICS OF THE NERVOUS SYSTEM AND MIND.

THE MBOUNDOU POISON.—The following are the principal points of a recent memoir by M. Testut on the mboundou poison of Gaboon, Africa, as given in a notice in *La France Médicale:*

In the first place, the author found the action of the poison varies according to the way it was administered, and the dose. With small doses there were invariably observed convulsive symptoms, indicating increased reflex excitability. If given in large doses, the animal appeared as if struck down, respiration was arrested in a few seconds, and the frog became a passive mass, not even responding, in any way whatever, to excitations; nevertheless, he is not dead; the heart continues to beat with normal regularity.

M. Testut therefore suspects that there may be two principles in this famous ordeal poison: one exciting the reflex activities, and the other stupefying and paralyzing them, this last acting only when large doses are given, and killing the animal before the convulsive phenomena have had time to appear. Whatever they or it may be, elimination takes place through the regular channels, and, in case of the frog, largely through the skin.

The abdominal lesions found after death from this poison, are of a congestive nature, and similar to those found after poisoning from strychnia and agaricus. These lesions, not being due to a direct or topical action, must be attributed to the vaso-motor centres in the spinal cord, disordered in their functions by the absorption of the poison. The mechanism of death from this poison in the higher animals, is, probably, asphyxia by respiratory arrest, a pulmonary anhæmatosis.

SPIRITUS NUCIS JUGLANDIS.—Dr. Edward Mackey, of Brighton, England (*Practitioner*, Dec.), gives the results of his experience in the treatment of obstinate vomiting, with spirit of walnut, a remedy almost obsolete, but for which he claims considerable efficacy as an anti-emetic, useful in many cases of obstinate emesis. The preparation he uses he obtained from Messrs. Southall, of Birmingham, and its formula is as follows:

Fresh walnuts, 30 oz.
Spirit of wine (rect.) 12 oz.; water q. s.
Distil 16 oz.

He has had good results from its use, in drachm doses, every one to four hours, in a little water, in cases of hysterical vomiting, the vomiting of obstinate dyspepsia, that of pregnancy, that due to anomalous causes, and even in cerebral vomiting. He has tried it also in septicæmia without effect; but this result is not surprising. He recommends its more extensive trial by the medical profession.

NITRITE OF AMYL.—Dr. Hugo Engel, *Phila. Med. Times*, Jan. 4, recommends inhalations of nitrite of amyl in cases of convulsions, when there is a general paleness of the whole surface, indicating almost a tetanus of the arterioles. In a case of very severe convulsions of a child, after the failure of bromides and chloral, and when heroic treatment was certainly indicated, he injected one-fourth of a grain of morphine into the forearm, and applied five drops of nitrite of amyl on a handkerchief to the nose, producing at once a deep sleep, that lasted eight hours. To be on the safe side, he injected one one-hundred and eightieth of a grain of atropia as an antidote to the morphine. A rapid recovery ensued, but treatment with the bromides was continued.

The use of the atropia, in this case, very probably saved the child's life from the effects of the morphia, which, considering its age—sixteen months,—was altogether too heroic, in our opinion, for anything but an almost hopeless case. It may, nevertheless, have had an indispensable part in producing the general result of the recovery. The case is a good illustration of the therapeutic use of the antagonism of these two agents.

TREPHINING IN EPILEPSY.—In the *Archives Génerale de Médicine* for December, 1878, Dr. Echeverria has published a *resumé* of the results of trephining in epilepsy, resulting from injuries of the skull. He has collected one hundred and forty-five cases of this operation. Of these, ninety-three were followed by recovery; eighteen by improvement; in five no change was produced; one was rendered worse; and death resulted in twenty-eight cases The causes of death in the fatal cases, were extremely varied, viz.: suppuration over the whole surface of the brain, hemorrhage into the brain, under the seat of operation; gangrene of the membranes and cerebral abscess; obstinate hemorrhage of the superior longitudinal sinus; meningitis and meningo-encephalitis.

Dr. Echeverria gives the following *resumé* of the results of his analysis of the cases referred to:

Trepanation is the best means which can be employed in the treatment of epilepsy caused by injuries of the skull.

The immediate operation appears to be almost as successful as the late; fever forms a serious contra-indication to the operation. Insanity and paralysis justify the operation.

Trephining succeeds equally well when syphilitic products upon the bones of the skull, and which have proved rebellious to specific treatment, act as the cause of epilepsy.

The success of the operation depends, in great part, upon our ability to prevent irritation of the cerebral meninges.

It is, finally, advisable to keep the patient under anti-epileptic treatment for some time after the operation, in order to overcome the so-called epileptic *habit* of the nervous system.—*N. Y. Med. Record.*

DIGITALINE.—The following are the conclusions of a memoir by Guido Cavazzini, *Ann. d'Omodei*, 1878, t. 245, p. 115. (Abstr. in *Bull. Gén. de Thérap.*)

1. In frogs the action of digitaline is shown with great effect on the heart, particularly on the ventricle, in exciting the muscular fibres, the degree being in proportion to the dose employed.

2. One or two drops of the solution, according to the season, accelerate the movements; six or seven cause tetanus of the ventricle.

3. Digitaline increases the tonicity of the cardiac fibres, and thus slows the repetition of the contractions, reducing their number very decidedly.

4. The auricles are only slightly or not at all excited by digitaline; in them the systolic frequence does not undergo any diminution analogous to that of the ventricles; this is why the ventricular tonicity embarrasses the cardiac functions.

5. The ventricular diastole never seems aroused, but appears to be subordinate to the action of the auricular muscular fibres. The auricles may become filled to excess, to the point of being considerably distended, and subsequently paralyzed, which excludes the idea of activity.

6. The assertion of some physiologists, that the myocardium retains its blood during the systole, cannot be admitted; its pallor, clearly observed, is a direct proof to the contrary.

7. Digitaline, proportionally to the time of the experiment and the quantity employed, accelerates the peripheral circulation according to the augmentation of the impulsive force of the heart. As the retardation of the ventricular contractions and the ventricular tetanus occurs, the circulation first diminishes, then is arrested.

8. The capillary net-work slightly dilates, and nevertheless the circulation may be accelerated, provided that the medicine does not oppose itself to the diastolic extension and the rhythmic frequency of the ventricle.

9. It appears, therefore, that the action of digitaline localizes itself principally on the heart, and acts only secondarily on the vessels.

10. Digitaline appears to increase the faculty of absorbing oxygen in the respiratory substance.

11. The opinion of the Berlin school that digitaline in small doses is excitant, and in large doses depressant, is not borne out by experience. This substance always excites the cardiac tonicity and dilates the vessels; in toxic dose it causes tetanus and rupture of the heart.

12. *En resumé*, digitaline has the effect of remedying feebleness of the cardiac systole; it may assist the peripheral circulation by augmenting the *vis a tergo* and dilating the capillaries; it may be of service in disorders accompanied by insufficient oxidation of the blood.

THE TREATMENT OF NEURALGIA.—Dr. E. C. Seguin, *N. Y. Med. Rec.* Jan. 4, narrates three cases of severe chronic trigeminal neuralgia, one of which was successfully treated with gelsemium and Fowler's solution, while the other two were not relieved until Duquesnel's aconitia, in gradually increasing doses, was employed. He closes his account as follows:

It seems to me that three conclusions may legitimately be drawn from the above related cases.

1. That there is a possibility of relief in most severe cases of epileptiform trigeminal neuralgia. The usually received opinion is that, in such cases, recourse must be had to operation upon deep branches of the nerve, excision of Meckel's ganglion, etc., and to the systematic use of morphia to make life endurable. After my experience with the above cases, I am dis-

posed to urge a sufferer from trigeminal neuralgia to make a trial of medicinal treatment.

2. The advantage of using medicines systematically. Not only should the doses of any one remedy be administered regularly and in progressively increasing doses, but several remedies should be used in succession, so as to profoundly affect the system. Of the medicines applicable to the treatment of neuralgia, the following are those which I can recommend most highly: aconitia, arsenic, iodide of potassium, gelsemium, belladonna, quinia, morphia, galvanism, the actual cautery, Thompson's solution of phosphorus.

3. In the treatment of chronic neuralgia and of many neuroses, it is necessary to obtain the physiological effects of the drug employed, in order to do good. This principle of heroic medication is one which ensures success in seemingly desperate cases, and its execution requires the utmost watchfulness on the part of the physician, and intelligence and faithfulness on the part of the patient and his attendants. Many unpleasant consequences of such treatment may be avoided if we at first give very small doses of the remedy, and then make a very progressive increase. The physiological effects are illustrated in the above cases; in the treatment of chorea by arsenic; of malarial affections by quinia; of spinal congestion and myelitis by belladonna; of syphilitic disease by mercury and iodide of potassium, etc., etc.

Inasmuch as the good effects noted in cases 2 and 3 were obtained by the action of Duquesnel's aconitia, it may not be amiss to close this short communication by quoting the conclusions of a report on aconitia, recently made to the N. Y. Therapeutical Society, by its committee on Neurotics.*

The chairman of this committee says:

"From the above cases, the following conclusions may be justly drawn, I think:

"1. The susceptibility of individuals to Duquesnel's aconitia varies enormously; one individual in the series having been severely affected by $\frac{1}{200}$ grain, while another tolerated with no special symptoms $\frac{1}{34}$ grain every three hours. On the average, distinct physiological and therapeutical effects were obtained by giving $\frac{1}{100}$ grain three times a day.

"2. Out of six cases of severe trigeminal neuralgia, one, probably a reflex neuralgia from a decayed tooth, was not at all benefited.

"Three cases, epileptiform in character, were slightly or only temporarily relieved. Two cases were cured. One of these had existed for seven years, with an interruption of twenty months, procured by resection of the affected nerve.

"It would thus appear that, while we cannot indorse Prof. Gubler's statement that Duquesnel's aconitia never fails, we must recognize in it one of the most powerful and best agents for relieving and curing trigeminal neuralgia.

"3. We do not as yet know the forms of trigeminal neuralgia which can be most influenced by aconitia."

STRYCHNIA.—Dr. R. Leared, in a paper read before the Medical Society of London, December 2, 1878 (*Brit. Med. Jour.*, Dec. 28) offers it as his opinion that a large class of cases of dyspepsia are due to nervous debility affecting the muscular apparatus of the stomach; indeed, he would divide all cases into those due to this cause and those owing to defective secretion. For this former class of cases he considers strychnia the foremost medicinal remedy; indeed, he says it is almost a specific for relaxation of the gastric muscular fibres.

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* *Vide N. Y. Medical Journal*, Dec., 1878, p. 621.

THE TREATMENT OF BASEDOW'S DISEASE.—Prof. Germain Sée (*La France Médicale*, Nov. 13) states that the only treatment he has seen succeed in exophthalmic goitre, is hydrotherapy combined with the administration of the tincture of veratrum viride. He says, "If the opinion I have given relatively to the paralysis of the vagi as the mechanism of the palpitations be the more correct one, the palpitations ought to give way under the influence of veratrum viride; and this is the case, the action of veratrum viride is comparable to that of digitalis, without the inconvenience of increasing vascular pressure. I prescribe the tincture to the amount of ten, then twelve, and finally up to twenty drops a day, divided into three or four doses, and continue this medication for many weeks or months. I have seen definitely cured by this method, (1) a young woman who had presented the three characteristic symptoms since fifteen years, and who had given up all treatment for three years; (2) a young woman attacked at the age of seventeen with palpitations and hypertrophy of the thyroid gland; (3) a case of exophthalmus with palpitations."

Dr. Sée's success with other methods has certainly been less than that of many others, and his wholesale skepticism ought not to be generally adopted by the profession. Almost every case of this disease is a study in itself, and routine therapeutics should have no place in its management.

ANÆSTHETICS.—At the meeting of the French Association for the Advancement of Sciences last year, M. Rabuteau read a paper on the action of anæsthetics, the conclusions of which, as given in *L'Union Médicale*, Oct. 15, are as follows:

1. The ethers have an action on vegetable organisms destructive to life; but the germinative property is not destroyed, though germination may be a little retarded.

2. The action of ether is different on cold and warm-blooded animals. If we put simultaneously under a bell glass a frog, a guinea-pig, and a sponge saturated with acetic ether, the guinea-pig survives, but the frog succumbs after a variable but always very short time. If we inject acetic ether under the skin of a guinea-pig there is no anæsthesia produced, but it occurs if we put him under a bell-glass. M. Rabuteau explains these facts as follows: the warm-blooded animal decomposes the ether while the frog does not; whence the division into two classes of the ethers: those that are modified in the organism and those that are not modified. It should be stated here, in passing, that white wine owes its heady properties to the ether it contains.

3. There are some ethers, the trichloracetates, that have the property of suppressing the sensibility without abolishing motion. They may have per-

haps a useful application in obstetrics. It is needful, nevertheless, to make a judicious selection, keeping in mind that the ethers of the ethylic alcohol are inoffensive.

Cerebral Accidents from Iodide of Potassium.—At the session of the Societè de Biologie, Jan. 4 (Rep. in *Progrès Méd.*), M. Hallopeau gave an account of the case of a syphilitic patient, thirty years of age, who had been for a long time under treatment with iodide of potash. He had first a purpura hemorrhagica followed by an alternate hemiplegia, which M. Hallopeau believed could be attributed to a hemorrhage due to the action of the iodide. Particulars not given.

Therapeutic Uses of the Bromides.—Rosenthal, in an article (*Wien. med. Presse*, 1878, No. 46) on therapeutics, speaks thus of the bromides:

Bromide of potassium, on account of the adynamia of the heart which it sometimes causes, must frequently be suspended. In addition to weakness, retardation, and irregularity of cardiac action, the bromide of potassium, in large and long-continued doses, may give rise to præcordial pain and mental disturbance. Bromide of potassium, although it occasionally improves the appetite to a marked degree, yet more frequently disturbs digestion when this is not originally strong. In such cases it should be taken at meal-times, or after draughts of milk, which preserves the stomach from irritation. *Bromide of sodium* is a milder preparation, more easily borne by the stomach; it is therefore preferable in the case of nervous women, of old people, and of children, since it has a salty taste, not in itself unwelcome to children. Rosenthal mixes the powdered bromide of sodium to the amount of ½ to 1 gramme (7½ to 15 grains) with bread-crumb, and thus smuggles it in, so to speak, as part of the food. He employs it in various nervous affections, as convulsions, laryngismus, nightmare, and disturbed sleep, giving it once or more daily. The chief objection to the bromide of sodium is its too ready deliquescence. Where enough cannot be given in the form of small pills, it may be given in powders, making up only enough for one or two days, or in solution with syrup.

Bromide of ammonium is a more stable preparation. It has a salty ammoniacal taste in solution, which may be disguised by syrup of orange-peel. Rosenthal has given it in doses of 6 or 8 grammes (ʒ iss. to ʒ ii.) daily, for months at a time, without mishaps. It acts well in epilepsy, whooping-cough, spasm of the glottis, and spastic laryngismus (in children as well as in hysterical persons), but no better than the other bromides. *Brominated camphor*, as appears from researches upon animals, affects the heart's action, and respiration, and the temperature, reducing them. Its formula is $C_{10} H_{15}$ Br. O, and it occurs in white crystals. It is not to be confounded with Laurent's "*bromkampher*," which occurs in red rhombic crystals, and the formula of which is $C_{10} H_{16} Br._2$ O. It is useful in the milder forms of epilepsy and in alcoholic trembling; but Rosenthal does not consider it equal to the bromides of potassium and sodium. As an antidote to strychnia it has proved valuable given in doses of 1 to 2 grammes (15 to 30 grains) twice or thrice, repeated at short intervals. In nervous palpitation of the heart, Rosenthal has given brominated camphor in the dose of .2 to .3 decigramme (3 to 4½ grains) several times a day, in *cachets du pain.* According to Berger, this salt is useful in sexual excitement and pollutions, but it fails in the more stubborn cases, where bromide of potassium, with opium, or tinct. verat. viridis (two or three drops on sugar until slowing of the pulse is attained), acts much more satisfactorily. In the case of a patient ordered small doses of brominated camphor on account of genital excitement, who took on his own responsibility 1 gramme (15 grains) at a dose, Rosenthal observed symptoms of weight and pressure in the head, shortness of breath, slowing of the pulse to sixty beats, weakness of the limbs, and mental disturbance to a marked degree, together with fear of death. Some ether upon sugar was administered, and later strong coffee, which soon dissipated the alarming symptoms. Rosenthal regards brominated camphor as particularly useful in irritation of the bladder. Both in that form due to taking cold and in that caused by the forcible retention of the urinary secretion, brominated camphor in the dose of .2 to .4 decigramme (3 to 6 grains) thrice daily quickly abated the annoying difficulty in urinating. In such cases it must be continued for some time. Sensitive persons experience fullness of the head (probably due to the action of the bromine); but this feeling leaves after a few doses. Long-continued use of brominated camphor sometimes causes derangement of the stomach, requiring suspension of the remedy. *Bromide of zinc* can be prescribed only in pill form or in solution, on account of its deliquescent properties. It is given at first in the dose of .1 decigramme (1½ grains) several times daily, and is gradually increased to .4 decigramme (6 grains). It is particularly useful in hysterical muscular spasm. Rosenthal does not consider this preparation superior to the bromide of potassium.

Hydrobromate of quinine is easily soluble in alcohol, but in water only in the proportion of 1 to 18, even when heated. It dissolves, however, in hot glycerine quite easily (1 to 4), and the solution remains clear for months. This can be diluted with water for hypodermic injection, until a syringeful contains only .1 decigramme (1½ grains) of the quinine salt. Rosenthal has used this with very good effect in a case of hysterical vomiting when opium had failed, the vomiting ceasing entirely after two or three days. Hydrobromate of quinine is also useful in the dose of ½ gramme (7½ grains) a day in general nervousness, in circumscribed headache, and in muscular cramp. Though recommended by Erlenmeyer in the painful paroxysms of locomotor ataxia, Rosenthal has found this preparation not to be depended upon. (*Philadelphia Med. Times,* Feb. 15, 1879.)

Brucia.—R. P. Robins, B. A., *Phil. Med. Times,* Feb. 15, in order to test the assertion of Klapp that strychnia fails to act on the motor nerves, and on the hypothesis that the contrary assertions of various authors were due to their having used impure samples of strychnia containing brucia, undertook a series of experiments upon the latter alkaloid, five of which are given. He used rabbits, dogs and frogs, injecting the brucia solution under the skin and testing the nerves with DuBois-Reymond's induction apparatus as modified by Helmholtz. The experiments, in fact, were identical with the strychnia experiments of Klapp in all but the agent employed, the solutions were of the same strength, and the methods the same. The result was that all the neurility was found impaired, the brucia alkaloid destroying the action of the motor nerves.

The author considers these results as corroborating those of Dr. Klapp in so far as they prove the motor-paralyzing action of brucia, and thus explain the action of strychnia alkaloids from which this has not been altogether eliminated.

Aconite —The following are the conclusions from an extended experimental inquiry into the physiological actions of aconite and aconitia, by Dr. G. Hunter Mackenzie, and published in the *Practitioner*, concluding in the number for March, 1879.

1. Aconite and aconitia act primarily on the respiration by their influence on the respiratory centre and peripheral sensory branches of the vagus.

2. They have no direct action on the heart, and only affect that viscus secondarily, through the medium of the lungs.

3. Their action on the nervous system consists in first, irritating, and secondly, paralyzing the peripheral sensory nerves and posterior roots of the spinal nerves. They increase the irritability of the peripheral motor nerves, and of the motor columns of the cord.

4. They do not induce muscular paralysis, but, on the contrary, increase the irritability of voluntary muscle.

5. They induce convulsions mainly through their augmenting the irritability of the anterior column of the cord, the motor nerves and muscles.

6. They firstly increase, and secondly diminish temperature.

7. Death ensues from asphyxia and respiratory collapse.

The following are some of the recently published articles on the Therapeutics of the Nervous System and Mind:

Ransohoff, Tetanus, Nerve-Stretching, Cure, *Lancet and Clinic*, Jan. 18.—Bartholow, Note on Some Points in regard to the Actions and Uses of Pilocarpine and its Salts, *Ibid*, Dec. 28.—Hayden, Notes on the Treatment of Chorea, *Dublin Jour. of Med. Sci.*, Jan.—Hack Tuke, Metalloscopy and Expectant Attention, *Jour. of Mental Science*, Jan.—Dufour, Note in regard to Functional Localizations in the Various Forms of General Paralysis, *Ann. Medico-Psychologiques*, Sept., 1878.—Neftel, Contribution to the Treatment of Neuralgias, *N. Y. Med. Record*, Feb. 1.—Devore, Note on Saturnine Hemiplegia and its Treatment with a Magnet, *Progres Med.*, Feb. 8.—Read, Chloral in the Treatment of Traumatic Tetanus, *Med. and Surg. Reporter*, March 1, 1879.—Gibney, Galvanism in the Treatment of Sciatica, *Am. Practitioner*, March.

BOOKS, ETC., RECEIVED.

Anatomie des Centres Nerveux. Par le Professeur G. Hugnenin (de Zurich). Traduit par le Dr. Th. Keller, Annoté par le Dr. Mathias Duval. Avec 149 figures intercalées dans le texte. Paris, 1879. 368 pages.

Traité de la Paralysie Générale des Aliénés. Par le Docteur Auguste Voisin. Avec XV. planche dessinées d' apres nature, lithographiées et coloriées. Graphiques, fac-simile. Paris, 1879. 540 pages.

Sulle Funzioni del Cervello. Ricerche sperimentali del Professori Luigi Luciani e Augusto Tamburini. Seconda Comunicazione Centri Psico-Sensori Corticali (Estratto *Dalla Rivista Sperimentale*, etc.). Reggio-Emilia, 1879. 78 pages.

Essai de Physiologie Générale appliquée a l'Étude de la Vie et de la Mort. Par le Dr. Élie Goubert. Paris, 1879. 132 pages.

Leçons sur la Physiologie Operative. Par M. Claude Bernard. Paris, 1879.

A Clinical Treatise on Diseases of the Liver. By Dr. Fried. Theod. Frerichs. In Three Volumes. Vol. I. Translated by Chas. Murchison, M. D., M. R. C. P. New York: Wm. Wood & Co., 1879.

Ueber die Folgen der Verletzungen auf Eisenbahnen, insbesondere der Verletzungen des Rückennmarks. Mit Hinblick auf das Haftpflichtgesetz. Dargestellt von Dr. Johannes Rigler. Berlin, 1879. 124 pages.

Die Furchen und Wülste am Grosshirn des Menschen. Zugleich als Erläuterung zu dem Hirnmodell. Von Ad. Pansch, Professor an der Universität Kiel. Mit drei lithographischen Tafelen. Berlin, 1879. 51 pages.

De l'Alcoolisme et de ses Consequences, au point de vue de l'etat physique, intellectuel et moral des populations. Par le Dr. E. Lancereaux. Paris, 1878. 41 pages.

The Transactions of the American Medical Association. Instituted, 1847. Vol. XXIX. Philadelphia. Printed for the Association, 1878. 1145 pages. (Including Prize Essay. Essays upon the Surgical Anatomy and History of the Common, External, and Internal Carotid Arteries, and the Surgical Anatomy and History of the Innominate and Subclavian Arteries, giving an Analysis of 173 Dissections of these Surgical Regions of the Neck, and a Collection of 1198 Cases in which the Results of Deligation are given, with other useful data. By John A. Wyeth, M. D.)

The Present Aspect of the Question of Tetanoid Paraplegia. By E. C. Seguin, M. D. (Reprinted from the *Archives of Medicine*, Feb., 1879.) New York: G. P. Putnam's Sons, 1879.

Report of the Department of Health of the City of Chicago for the Year 1879.

American Clinical Lectures: The Diagnosis of Progressive Locomotor Ataxia. By E. C. Seguin, M. D. New York: G. P. Putnam's Sons, 1878.

Clinical Lecture on Syphilitic Brain-Lesions. By E. C. Seguin, M. D. (Reprinted from the *New York Medical Journal*, Sept., 1878.)

A Contribution to the Medicinal Treatment of Chronic Trigeminal Neuralgia. By E. C. Seguin, M. D. (Reprinted from *The Medical Record*, Jan. 4, 1879.) New York, 1879.

Report on Aconitia in Trigeminal Neuralgia. By E. C. Seguin, M. D. (Reprinted from the *New York Medical Journal*, Dec., 1878.) New York: D. Appleton & Co., 1878.

Due Casi di Localizzazione Cerebrale. Per Dottori D. Maragliano e G. Seppilli. (Estratto *Dalla Rivista Sperimentale di Freniatria e Medicina Legale*, 1878.)

A Clinical Lecture on Anæsthetic Leprosy. By James Nevins Hyde, M. D. (Reprinted from the *American Practitioner*, February, 1879.)

The Antiquities and Platycnemisin of the Mound Builders of Wisconsin. By J. M. De Hart, M. D.

Report of Investigations into the Pathogeny of Diphtheria. Conducted by Edward Curtis, M. D. and Thomas E. Satterthwaite, M. D. New York, 1877.

The Causes of Sudden Death of Puerperal Women. An address in Obstetrics and Diseases of Women and Children. (Delivered before the American Medical Association, June 5, 1878.) By Edward W. Jenks, M. D. (Extracted from the Transactions of the American Medical Association.) Philadelphia, 1878.

The Nature and Diagnosis of Neurasthenia (Nervous Exhaustion). By Geo. M. Beard, M. D. (Reprinted from the *New York Medical Journal*, March, 1879.) D. Appleton & Co. New York, 1879.

The Evidence of the Senses—Inaugural Address. By W. G. Stevenson, M. D., President of the Poughkeepsie Society of Natural Science. 1878.

Electricity in its Relations to Medicine and Surgery. Its Application in Neuralgia, Chorea, Hysteria, Spinal Irritation, Epilepsy, Exophthalmic Goitre, Torticollis, etc. By A. D. Rockwell, M. D. (Reprint from *Virginia Medical Monthly*, February, 1879.)

Aphasia, or Aphasic Insanity, Which? A Medico-Legal Inquiry. By Dr. C. H. Hughes, St. Louis, Mo. (From the *American Journal of Insanity*, January, 1879.)

The Value of Absent "Tendon-Reflex" as a Diagnostic Sign in Locomotor Ataxia. With an Analysis of Eight Cases. By A. McL. Hamilton, M. D. (Reprint from the *Boston Medical and Surgical Journal*, December 19, 1878.) Cambridge, Riverside Press, 1879.

Are Inebriates Automatons? By George M. Beard, M. D., of New York. Read before the American Association for the Cure of Inebriates, at Boston. October, 1878.

Diphtheria, its History, Symptoms, Causes, Prevention and Cure.

Scarlet Fever and its Successful Treatment. By Melville C. Keith, M. D Lincoln, Nebraska. 1879.

A Case of Ovariotomy (Successful). By Edward Borck, M. D. (Reprinted from the *St. Louis Medical and Surgical Journal*, April, 1878.) St. Louis, 1879.

Puerperal Laceration of the Cervix Uteri and the Operation of Trachelorraphy, as a Means of Cure. By E. C. Dudley, M. D. (Reprinted from the *Chicago Medical Journal and Examiner*, March, 1879.)

Fifty Years Ago. An Address to the Graduating Class of the Medical College of the Pacific, for 1878. By Henry Gibbons, Sr., M. D.

Address of W. O'Daniel, M. D., President of the Medical Association of Georgia. (Reprint from the Transactions of the Medical Association of Georgia.) 1878.

In Memoriam, Dr. Landon R. Longworth. By F. Forchheimer, M. D.

Transactions of the Detroit Medical and Library Association. January, 1879, Detroit.

Fifth Biennial Report of the Board of State Commissioners of Public Charities of the State of Illinois. Springfield, 1878.

Transactions of the Medical Society of the State of Tennessee at its Forty-fifth Annual Meeting. 1878.

The Metric System. By David Prince, M. D. From *American Practitioner*, December, 1878.

Sixth Annual Report of the Northern Hospital for Insane of the State of Wisconsin, for the Fiscal Year ending September 30, 1878.

First Annual Report of the Health Department of the City of St. Louis, under the Provisions of the New Charter, for Fiscal Year ending April 10, 1878. Charles W. Francis, Health Commissioner.

Circular of Information Relating to the Pennsylvania Training School for Feeble-Minded Children. Media, Pa., 1879.

Twenty-sixth Annual Report of the Pennsylvania Training School for Feeble-Minded Children. Media, Pa., 1878.

Extracts from Twentieth Annual Report of Commissioners of Lunacy of Scotland, for the year 1877, with an Introduction by H. B. Wilbur, M. D.

Thirteenth Report of the Board of Trustees of the Connecticut Hospital for the Insane. 1879.

Reports of the Trustees and Superintendent of the Butler Hospital for the Insane, presented to the Corporation at their Annual Meeting, January 22, 1879.

Report of the Pennsylvania Hospital for the Insane, for the Year 1878.

Nineteenth Annual Report of the Medical Superintendent of the State Asylum for Insane Criminals, for the Year ending September 30, 1878.

Twenty-fourth Annual Report of the Dayton Asylum for the Insane, for the Year 1878. Ohio.

Fifth Biennial Report of the Illinois Northern Hospital for the Insane at Elgin. October 1, 1878.

Report of the Pennsylvania Hospital for the Insane, for the Year 1878.

Tenth Annual Report of the Willard Asylum for the Insane for the Year 1878.

Maternal Impressions; Mothers' Marks. An Expose of a Popular Fallacy. By Roswell Park, A. M., M. D. (Reprint from *The Southern Clinic*, February, 1879.)

THE FOLLOWING FOREIGN PERIODICALS HAVE BEEN RECEIVED SINCE OUR LAST ISSUE.

Allgemeine Zeitschrift fuer Psychiatrie und Psychisch. Gerichtl. Medicin.
Annales Médico-Psychologiques.
Archiv fuer Anatomie, Physiologie, und Wissenschaftl. Medicin.
Archiv fuer Path. Anatomie, Physiologie, und fuer Klin. Medicin.
Archiv fuer die Gesammte Physiologie der Menschen und Thiere.
Australian Practitioner.
Brain.
British Medical Journal.
Bulletin Générale de Thérapeutique.
Centralblatt f. d. Med. Wissenschaften.
Centralblatt f. d. Nervenheilk., Psychiatrie, etc.
Cronica Med. Quirurg. de la Habana.
Dublin Journal of Medicine and Surgery.
Deutsche Medicinische Wochenschrift.
Edinburgh Medical Journal.
Gazetta Frenocomia di Reggio.
Gazetta Medica de Roma.
Gazette des Hopitaux.
Gazette Médicale de Bordeaux.
Glasgow Medical Journal.
Hygeia.
Hospitals Tidende.
Journal de Médicine et de Chirurgie Pratiques.
Journal of Mental Science.
Journal of Psychological Medicine.
Journal de Medecine de Bordeaux.
La France Médicale.
Lancet.
Le Progrès Medical.
Lo Sperimentale.
L'Union Medicale.
Mind.
Nordiskt Medicinskt Arkiv.
Norsk Magazin for Lagensvidenskabens.
Psychiatrisches Centralblatt.
Rivista Clinica di Bologna.
Rivista Sperimentale di Freniatria e de Medicina Legale.
Revue Medicale du Nord-Est
Revue Mensuelle de Medicine et de Chirurgie.

Schmidt's Jahrbuecher der In- und Auslandischen Gesammten Medicin.
St. Petersburger Med. Wochenschrift.
The Practitioner.
Upsala Lakareformings Forehandlinger.

The following domestic exchanges have been received:

American Journal of Insanity.
American Journal of Medical Sciences.
American Journal of Obstetrics.
American Journal of Pharmacy.
American Medical Weekly.
American Practitioner.
Archives of Dermatology.
Atlanta Medical and Surgical Journal.
Boston Medical and Surgical Journal.
Buffalo Medical Journal.
Canada Medical Record.
Canadian Journal of Medical Sciences.
Chicago Medical Journal and Examiner.
Clinic.
Cincinnati Lancet and Observer.
Detroit Lancet.
Index Medicus.
Maryland Medical Journal.
Medical Brief.
Medical News and Library.
Medical Record.
Medical and Surgical Reporter.
Michigan Medical News.
Nashville Journal of Medicine.
New Remedies.
New York Medical Journal.
New York Med. and Surg. Brief.
Ohio Medical and Surgical Journal.
Pacific Medical and Surgical Journal.
Proceedings of Med. Society of the County of Kings, N. Y.
Pharmacist.
Philadelphia Medical Times.
Quarterly Journal of Inebriety.
Richmond and Louisville Medical Journal.
Southern Practitioner.
Southern Clinic.
St. Louis Medical and Surgical Journal.
St. Louis Clinical Record.
Toledo Medical Journal.
Virginia Medical Monthly.

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Original Articles, Selections and Translations.

ART. I.—SAPONIN IN ITS RELATIONS TO PHYSIOLOGY.

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(*Continued from page* 291.)

GENERAL ACTION.

UNDER this head will be studied the action of the drug when introduced by absorption, and when it is injected into the blood-vessel system.

ACTION ON THE NERVOUS SYSTEM.

The peripheral *afferent* and *efferent nerves* remain unaffected when saponin is introduced into the veins of the general circulation. The excitability of the nerves for electrical irritants remains unaffected under these circumstances. When introduced into an artery, such as the femoral, the nerves of the part of the body into which the drug was injected are first excited and then paralyzed by the drug. The other nerves of the body retaining their functions for a long period thereafter, proves, as was before stated, that this effect is due to the local action of the drug.

On the *spinal cord* the drug has a more decided action. After the destruction of the brains of frogs it is well known that spinal reflexes become more marked. When, however, frogs thus prepared are poisoned with saponin, a gradual abolition of reflex action occurs. This was determined by the use of a solution of acetic acid which was just sufficient in the normal animal to produce reflex movements when applied to the web of the feet. About a half hour after the poisoning the solution of the acid previously employed had to be increased in strength in order to produce reflex movements. After a varying period—usually about an hour—it was found that application of strong sulphuric acid failed to produce these movements.

The same results, less perfectly, it is true, were obtained in dogs, rabbits, and cats in whom section of the pons had been made, at its junction with the medulla oblongata. Saponin therefore produces abolition of the reflex activity of the spinal cord. This paralysis is not due to any action on the motor nerves, as these respond to irritants for some time after the cessation of reflex movements.

The general action of saponin on the *brain* is very interesting, as it appears to be a sleep-producing agent. .02–.1 gram. of the drug injected into a mesenteric or femoral vein of a cat produces deep sleep in 15–25 minutes, from which the animal can be aroused, to fall again into the same somnolent state when the excitation is removed. If the dose be very large the animal gradually loses all sensibility and falls into a state of coma, from which it cannot be aroused, and dies in about two hours. If a smaller dose be given a cat or a dog this state of insensibility lasts several hours, when the animal gradually recovers without any untoward symptoms. An injection of .002 into the back of a mouse, pigeon, or sparrow, also produces this sleep, accompanied by loss of sensibility and followed by coma and death.

Accompanying and preceding this somnolent state in mammals, and replacing it in frogs, there is complete abolition of all the so-called voluntary movements. "Saponized" frogs when placed on their backs make no attempt to re-attain the abdominal position, though at the time the reflex activity may be unaffected, showing that either the animal is unaware of its position, owing to an action of the drug on the perceiving organs of the central nervous system; or that there is paresis of the peripheral sensory or motor nerves. As reflex movements can still be excited the latter cannot be the cause.

Saponin therefore *produces sleep by greatly reducing or even abolishing the perceptive faculties of animals.*

When introduced into the general circulation of mammals and frogs, saponin exerts no influence whatsoever on the functions of the *muscles.*

ACTION ON THE CIRCULATORY SYSTEM.

On the blood-pressure in the Arteries.—Hoppe* saw a weak solution of saponin first excite and then depress the actions of the heart.

Köhler† found in rabbits and dogs, that after an evanescent rise the blood-pressure always diminishes, and believes this to be due to excitation, followed by paralysis, of the vasomotor "centres." He further says that in the "saponized" animal the heart is cut off from all nervous influences except those exerted by its own intrinsic gangliæ.

For reasons which will be apparent I will divide my experiments on the circulatory action of saponin into the following three classes:

1. The action of saponin on the arterial pressure when injected into a jugular vein;
2. The action of saponin on the arterial pressure when injected into a femoral vein;
3. The action of saponin on the arterial pressure when introduced into an efferent blood-vessel.

Saponin injected into a jugular vein.—Owing to the heart-nerves and blood-vessels varying in the different species of mammals, I have endeavored to separate the experiments on the different animals as much as possible, and at the same time to keep up a comparison between them. In the first series of experiments the only operations were the insertion of a canula, which was connected with a manometer,* into the common carotid or femoral arteries, and then placing a canula into the external jugular vein through which the solution of the drug was injected towards the heart.

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* "Die Nervenwirkung d. Arzneimittel," Hft. IV., p. 137.

† *Arch. f. exp. Path.*, I., p. 138-162. "Die locale Anæsthesir. d. Saponin," p. 78. *Physiologischen Therapeutik*, 1876, p. 286.

Following such an injection in rabbits I never saw a rise but always a very decided fall in the arterial pressure, as will be illustrated in the following experiments:

No. of Experiment.	Animal.	Time.	Pressure.	REMARKS.
1.	Albino Rabbit.	Oct. 4, 1877.		Canula in carotid artery.
		9.34.	71.9 mm.	
		.10	71.9 "	
		.20	71.9 "	
		.30	71.9 "	
		.40	71.8 "	
		.50		Injected .05 gram. into external jugular vein towards the heart.
		.53	71.9 "	
		9.35.	66.3 "	
		.10	40.9 "	
		.20	40.7 "	
		.40	46.3 "	
		.50	54.9 "	
		9.36.	55.7 "	Animal died about this time.

In the cat the fall in the blood-pressure produced by the injection of saponin into the jugular vein is followed by a secondary rise, so that the pressure resulting may greatly exceed the normal arterial pressure.

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* This consisted of a U-shaped tube containing mercury. On this mercury was placed a swimmer of hard rubber and attached to this was a wire which wrote the variations of pressure on a revolving drum previously sooted with lamp-black.

No. of Experiment.	Animal.	Time.	Pressure.	REMARKS.
3.	Cat.	Nov. 4, 1877.		Canula in carotid artery.
		3.21.	99.4 mm.	
		.10	99.4 "	
		.20		Injected .037 gram. into external jugular vein towards the heart.
		.30	99.4 "	
		.40	96. "	
		.50	89.7 "	
		3.22.	87.5 "	
		.10	89.1 "	
		.20	92.2 "	
		.30	95.1 "	
		.40	97.9 "	
		.50	99.4 "	
		3.23.10	108.4 "	

When, however, a very large dose of the drug was injected into the jugular vein of a cat this secondary rise in arterial pressure did not occur.

No. of Experiment.	Animal.	Time.	Pressure.	REMARKS.
4.	Cat.	Dec. 5, 1877.		Canula in carotid.
		1 sec.	83.9 mm.	
		5 "		Injected .2 gram. into external jugular vein towards the heart.
		10 "	78.4 "	
		20 "	76.8 "	
		30 "	76.1 "	
		40 "	57.4 "	
		60 "	28.7 "	
		100 "	00. "	The animal is dead.

In the dog the same decided fall in pressure is observed to follow the introduction of a large dose of saponin into the jugular vein. This is however always followed by a secondary rise in the arterial pressure.

To what is the fall in the arterial pressure due? A diminished blood-pressure may be due: first, to a paralysis of the vaso-motor "centres" allowing a large quantity of blood to accumulate in the capillaries; second, it may be due to an active dilatation of the walls of the blood-vessels through the agency of the vaso-dilator nerves; third, to irritation of the vagus centres, trunks, or peripheral extremities; fourth, to a contraction of the pulmonary blood-vessels preventing the ærated blood from having free access to the arterial heart; fifth, to a paralysis of the heart itself.

It was first shown by Stilling* and Henle,† that certain nerves have for their function the contracting of the walls of the blood-vessels. To these the name of vaso-motors and vaso-constrictors has been bestowed. In 1859, Schiff,‡ and in 1871, Owsjannikow§ experimentally demonstrated that these nerves were under the control of a "centre" situated in the medulla oblongata, to which the name of "vaso-motor centre" was given. It is now well known to physiologists that irritation of a sensory nerve frequently‖ produces, through reflex stimulation of this "centre," a decided rise in the blood-pressure. This rise is by no means certain to occur if the pneumogastrics remain intact, as under these circumstances I have observed that irritation of the anterior crural nerve produces a very decided fall in the pressure, which was replaced by a rise when the pneumogastrics were cut or their inhibitory fibres paralyzed by atropine.

It was therefore necessary in the experiments on saponin to first cut the pneumogastric nerves; then to irritate the anterior crural, and observe the rise in the pressure produced in the manometer connected with the carotid or femoral arteries of the animal experimented upon. The drug being now given it was possible to determine if the vaso-motor "centre" was affected, by observing whether or not the irritation of a sensory nerve still produced an increased blood-pressure.

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* *Unters. u. d. Spinal-Irritation*, Leipzic, 1840, p. 163.

† *Path. Untersuch.*, Berlin, 1840, p. 105.

‡ *Unters. z. Physiol. d. Nervensystems*, Frankfort, 1855, p. 219.

§ *Ludwig's Arbeiten a. d. phys. Laborat.*, Leipzic, VI., p. 21.

‖ After curarization the rise in the blood-pressure from irritation is almost constant.

No. of Experiment.	Animal.	Time.	Pressure.	REMARKS,
6.	Dog.	Dec. 17, 1877.		Canula in carotid. Pneumogastrics cut.
		1 sec.	121.5 mm.	
		5 "		Injected .3 gram. into internal jugular vein.
		10 "	113.7 "	
		20 "	104.3 "	
		22 "		Irritation of anterior crural nerve.
		23 "	130.9 "	
		25 "	138.7 "	Cessation of the irritation.
		27 "	105.1 "	
7.	Cat.	Dec. 17, 1877		Canula in carotid artery. Pneumogastrics cut.
		1 sec.	80.6 mm.	
		5 "		Injected .25 gram. into internal jugular vein.
		10 "	62. "	
		12 "		Irritation of anterior crural nerve.
		13 "	67.5 "	
		17 "	74.7 "	
		20 "	79.2 "	
		21 "		Cessation of irritation.
		24 "	58.8 "	

No. of Experiment.	Animal.	Time.	Pressure.	REMARKS.
8.	Albino Rabbit.	Oct. 8, 1877.		Canula in carotid. Pneumogastrics cut. Injected .015 gram. into external jugular vein.
		1 sec.	78.1 mm.	
		5 "		Irritated sciatic nerve.
		7 "	100. "	
		15 "	109.2 "	
		20 "	113.8 "	
		21 "		Cessation of irritation.
		23 "	54.8 "	

It will be observed in the foregoing experiments that irritation of a sensory nerve still produces a rise in the pressure even if very large doses of saponin have been given. The cause of the diminished blood-pressure is therefore not to be sought for in a paralysis of the vaso-motor "centre" or nerves, as no such paralysis exists in animals poisoned by saponin.

The same result was arrived at through the following experiments where the greater portion of the vaso-motor nerves were cut, through high section of the spinal cord, after which operation saponin produced a fall in the pressure.

No. of Experiment.	Animal.	Time.	Pressure.	REMARKS.
9.	Cat.	Dec. 10, 1877.		Canula in carotid. Cord cut at fifth cervical vertebra.
		1 sec.	98.4 mm.	
		4 "		.1 gram. into external jugular vein.
		10 "	97.8 "	
		15 "	87.2 "	
		20 "	87.2 "	
		25 "	93.1 "	
		30 "	96.3 "	
		35 "	98.4 "	
		40 "	98.4 "	
		120 "	98.4 "	

No of Experiment.	Amount.	Time.	Pressure.	REMARKS.
11.	Dog.	Dec. 6, 1877.		Canula in carotid. Cord cut at fifth cervical vertebra.
		1 sec.	137. mm.	
		6 "		.3 gram. into internal jugular vein.
		10 "	137. "	
		12 "	133.2 "	
		15 "	131.1 "	
		20 "	131. "	
		30 "	129.5 "	
		40 "	121.6 "	
		50 "	128.7 "	
		55 "	135.6 "	
		60 "	137. "	
		120 "	137. "	

The experiments of various physiologists, and especially those of Schiff[*] and Goltz,[†] have taught us that, while there are vaso-constrictors, there are also vaso-dilating nerves. Comparatively little is known of these, and, by some physiologists, their existence is still doubted. Consequently it is difficult at the present time to determine whether or not the fall in the blood-pressure is caused by their being irritated by saponin. All that I have been able to do thus far to determine if the fall in the blood-pressure following an injection of this drug into the jugular vein is due to such an irritation, has yielded negative results. If such an active dilatation of the capillaries takes place it must be observable in the vessels in the web of the frog's foot, but, so far as my observations have gone, no such effect ever followed the injection of the drug into the lymph sacs of this animal. On the contrary the size of the capillaries diminishes.

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[*] *Mitth. d. naturforsch. Gesellsch. in Bern*, 1856. *Oversigt over det kgl. danske Vidensk. Selsk. Forhandlunger*, 1857, Nr. 8. *Comptes Rendus*, 1862, II., p. 540, etc.

[†] *Arch. f. d. ges. Physiologie*, IX., p. 174, 197. 1874.

Were the diminished arterial pressure due to irritation of the pneumogastric centres or trunk, it would cease to occur when the connections of this nerve with the heart are destroyed. This was done in a number of experiments by cutting the neck pneumogastrics. If now on injecting saponin, the pressure still diminished, it would be conclusive proof that irritation of these structures was not the cause of the fall.

No. of Experiment.	Animal.	Time.	Pressure.	REMARKS.
12.	Albino Rabbit.	Nov. 15, 1877.		Canula in carotid artery. Pneumogastrics cut.
		12. 04.	61.1 mm.	
		.10		.015 gram. into external jugular vein towards the heart.
		.20	59.5 "	
		.30	34.3 "	
		.40	31.1 "	
		.50	37.7 "	
		12.06	59.4 "	Animal died some hours later.

As the fall of pressure, in these and in a large number of similar experiments on rabbits, still occurred even when the pneumogastrics were cut, it should perhaps be concluded that this fall was independent of any action on these nerves. But before doing this it will be well to see whether the same holds good in dogs and cats.

No. of Experiment.	Animal.	Time.	Pressure.	REMARKS.
15.	Large Cat.	Dec. 3, 1877.		Canula in carotid. Vagi cut.
		1 sec.	102.6 mm.	
		7 "		.023 gram. into external jugular vein.
		9 "	93.2 "	
		12 "	92.1 "	
		15 "	81.6 "	
		25 "	60.4 "	
		30 "	67.6 "	
		40 "	79.5 "	
		45 "	86.4 "	Animal recovered.

From these experiments it appears that the pneumogastrics have nothing to do with the fall in blood-pressure in cats.

No. of Experiment.	Animal.	Time.	Pressure.	REMARKS.
16.	Small Dog.	Nov. 13, 1877.		Canula in carotid. Vagi cut in neck.
		12.44.	95. mm.	
		.10		Injected .16 gram. into jugular vein towards the heart.
		.20	95. "	
		.30	104.1 "	
		12.45	95.2 "	
		.10	100.4 "	
		12.46.20	103.3 "	Animal recovered.
17.	Hound.	Nov. 22, 1877.		Canula in carotid. Vagi cut.
		1 sec.	138.8 mm.	
		3 "		Injected .25 gram. into external jugular vein towards the heart.
		4 "	139.3 "	
		11 "	149.2 "	
		15 "	152. "	
		24 "	144.9 "	
		60 "	137. "	Animal recovered.

From these last experiments on dogs we arrive at a conclusion which is apparently at variance with the results obtained in rabbits and cats. Why is it that in rabbits and cats, the blood-pressure is still diminished by the drug when the vagi are cut, yet in dogs a rise in the pressure occurs under the same circumstances? It may be due to an anatomical difference in the distribution of the pneumogastric fibres in different animals. In the cat and rabbit, as was shown by Ludwig and Cyon,* and others, there is a little nerve lying close to the main vagus trunk in the neck, of which it is a branch, which has the function of a true circulatory depressor; *i. e.*, when this nerve is cut and its central end irritated, the pressure descends though the frequency of the pulse remains unaffected.† In the dog this little nerve is included in the main vagus trunk, and when the latter is cut the depressor nerve is also cut. In the rabbit, and frequently in the cat, when the vagi trunks are cut the depressors escape. It was therefore necessary to determine if the same rise in pressure as is produced in dogs by saponin, after the vagi trunks have been cut, occurs also in cats and rabbits when the depressors were cut with the vagi trunks.

No. of Experiment.	Animal.	Time.	Pressure.	REMARKS.
18.	Rabbit.	Nov. 19, 1877.		Canula in carotid. Vagi and depressors cut.
		1 sec.	86.1 mm.	
		5 “	85.9 “	
		10 “		Injected .062 gram. into jugular vein.
		11 “	84.2 “	
		13 “	69.6 “	
		17 “	64.8 “	
		20 “	62. “	In three minutes the animal was dead.
20.	Large Cat.	Dec. 3, 1877.		Canula in carotid. Vagi and depressors cut.
		1 sec.	111.6 mm.	
		4 “		
		7 “	103.9 “	.03 gram. into external jugular vein.
		12 “	98.4 “	
		16 “	92.5 “	
		17 “	108.2 “	
		20 “	112.7 “	
		24 “	118.5 “	
		26 “	122. “	
		40 “	120.9 “	

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* *Bericht d. Sächs. Gesellsch.*, 1866.

† To prevent reflex inhibition of heart's movements in these experiments the vagi nerves must previously be cut.

The depressor nerves being cut in these animals and saponin still producing a diminished blood-pressure, some other cause must be sought to explain the rise in pressure following the injection of the drug into the jugular veins of dogs in whom the pneumogastrics have been cut.

On referring to the experiments on dogs, where a fall in the blood-pressure occurred, it will be observed that the solution of saponin was introduced into the internal jugular vein. In order to keep the injecting canula in the vein, a canula over four inches long was used, while in the experiments where the pneumogastrics were cut, the drug was injected by means of an ordinary hypodermic needle tied into the external jugular vein. By the former mode of introduction we have the direct action of the drug on the heart, which produces the lowering in the pressure, while by the latter method the drug only reaches the heart muscle after being diluted with the blood coming from other portions of the body. That this explanation is the correct one will be seen in the following experiments on dogs, where, after the pneumogastrics were cut, and saponin injected into the heart, a fall instead of a rise in blood-pressure was produced.

No. of Experiment.	Animal.	Time.	Pressure.	REMARKS.
22.	Large Dog.	Nov. 18, 1877.		Canula in carotid. Vagi cut. A 4 in. long injecting canula introduced into the internal jugular vein towards the heart.
		1 sec.	90. mm.	
		3 "		.125 gram. into heart.
		5 "	84.4 "	
		11 "	80.7 "	
		40 "	75. "	
		2 min.	91. "	
24.	Small Dog.	Dec. 8, 1877.		Canula in femoral artery.
		1 sec.	96.4 mm.	
		3 "		.1 gram. into heart.
		10 "	86.2 "	
		15 "	81.3 "	
		20 "	79.8 "	
		25 "	85.2 "	
		30 "	86.7 "	
		40 "	97.6 "	

The reason why an injection of saponin into the external jugular vein of rabbits and cats produces a fall becomes apparent after these experiments. In these smaller animals, owing to the shortness of the distance from the point of injection to the heart, comparatively little dilution of the injected material occurs, before its effects on the heart become manifest, and the same result is obtained as from an injection directly into the hearts of dogs. When, however, saponin is injected peripherally into the external jugular vein in the smaller animals, similar phenomena to those which follow an injection into the external jugular of dogs occur.

No. of Experiment.	Animal.	Time.	Pressure.	REMARKS.
25.	Rabbit.	Nov. 19, 1877.		Canula in carotid.
		1 sec.	75.7 mm.	
		6 "	75.7 "	
		10 "		Injected .02 gram. into external jugular vein towards the periphery.
		12 "	75.7 "	
		14 "	78. "	
		16 "	79.5 "	
		17 "	81.4 "	
		20 "	80.2 "	
		24 "	79. "	
		60 "	59.7 "	

These experiments lead naturally to the conclusion that the fall in blood-pressure following an injection of saponin into the jugular vein is due to the direct action of the drug on the heart. What is the nature of this action? Is it on the nerves or ganglia, or on the muscular tissue?

That the fall in blood-pressure is not due to any action on the heart nerves is shown by the circumstance that a "saponized" heart will contract when mechanically irritated. Besides this, in about two-thirds of the experiments, this muscle continued to contract "automatically" for a long time after the respirations had ceased; but, though the left ventricle was filled with liquid blood, none of it was expelled into the aorta by these contractions. On closer examination it was observed that while the external muscular fibres of the heart still continued to contract, the internal fibres were absolutely motionless. Microscopically examined, the internal muscular fibres were found to have lost their striations and to present changes like those previously described as being produced by the local action of saponin on the skeleton muscles. In the experiments where the drug produced death in a few seconds these changes were best marked, and the hearts of these animals could no longer be brought to contract by irritation.

The rise in the arterial pressure succeeding the fall will be treated of later.

Action of saponin on the blood-pressure when injected into a femoral vein.—Experiments on rabbits. The first thing noticeable in these experiments is that doses which were immediately fatal when introduced into the jugular vein could be injected with impunity into the femoral vein. This is another argument in favor of the view before expressed: that, by the former method of injection, direct paralysis of the heart is produced. The arterial pressure is augmented by the latter method of introduction.

No. of Experiment.	Animal.	Time.	Pressure.	REMARKS.
28.	Albino Rabbit.	Dec. 14, 1877.		Canula in carotid.
		1 sec.	87.3 mm.	
		5 "		.035 gram. into femoral vein.
		7 "	87.6 "	
		15 "	88.1 "	
		30 "	88.1 "	
		50 "	89.6 "	
		60 "	89.7 "	
		70 "	89.9 "	
		75 "	88.4 "	
		90 "	87.6 "	
		120 "	82.1 "	

The same augmentation in the blood-pressure was observed in the experiments on cats and dogs.

The arterial pressure can be augmented by a drug :

1. By paralysis of the vagus roots, trunks or terminal heart fibres;
2. By stimulation of the afferent (sympathetic) circulatory fibres;
3. By stimulation of the vaso-motor centres or fibres;
4. By direct irritation of the walls of the blood-vessels.

Is the increased blood-pressure due to paralysis of the vagus? From the experiments which I made to determine the action of saponin on the cardio-inhibitory nerves, it appeared at first as if this was the true explanation of the augmented pressure. To determine this more certainly, I cut the pneumogastrics in a number of animals, and then observed the blood-pressure after an injection of the drug into the femoral vein.

No. of Experiment.	Animal.	Time.	Pressure.	REMARKS.
32.	Rabbit.	Dec. 11, 1877.		Canula in carotid. Vagi cut.
		1 sec.	57.4 mm.	
		5 "		.015 gram. into femoral vein.
		13 "	59.8 "	
		20 "	59. "	
		30 "	59.6 "	
		40 "	59.2 "	
		50 "	59.6 "	
		60 "	61.4 "	
		65 "	61.7 "	
		120 "	57.4 "	
34.	Dog.	Nov. 13, 1877.		Canula in carotid. Vagi cut.
		3 09	123.1 mm.	.15 gram. into femoral vein.
		.10	131.4 "	
		.20	125. "	
		.30	124.7 "	
		.40	128.9 "	
		.60	127.8 "	

On referring back to experiments 17 and 20 it will be seen that a rise in the blood-pressure can follow an injection into the jugular vein, notwithstanding the previous section of the pneumogastrics. As, following the injection into a femoral vein, an augmentation in the arterial pressure is produced even after the previous section of the vagi, it is conclusively demonstrated that the rise in pressure is not due to their paralysis.

To determine whether this augmentation is due to stimulation of the vaso-motor "centre." I cut the spinal cord in rabbits, cats and dogs, at the fifth cervical vertebra,* in order to cut the larger number of the vaso-motor nerves, and then introduced saponin into the femoral vein. Under these circumstances the blood-pressure never rose.

No. of Experiment.	Animal.	Time.	Pressure.	REMARKS.
35.	Albino Rabbit.	Dec. 20, 1877.		Canula in carotid. Spinal cord cut at lower edge of fifth cervical vertebra.
		1 sec.	76.7 mm.	
		3 "		.035 gram. into femoral vein.
		10 "	75.8 "	
		20 "	71.6 "	
		30 "	69.5 "	
		40 "	66.1 "	
		60 "	63.6 "	
37.	Large Dog.	Dec. 12, 1877.		Canula in carotid. Medulla oblongata destroyed. Artificial respiration.
		1 sec.	122.to 122.5 mm	
		5 "		.1 gram. into femoral vein.
		10 "	122.to 122.5 "	
		15 "	122.to 122.5 "	
		20 "	122.to 122.5 "	
		40 "	122.to 122.5 "	
		60 "	122.to 122.5 "	
		120 "	122.to 122.5 "	In ten minutes the pressure was still the same.

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* That is, above the origin of the splanchnics.

It was found in a large number of experiments similar to the above, that the rise in pressure never occurred when the medulla oblongata was destroyed at the calamus, and that it was reduced to a minimum by section of the spinal cord in the neck. By the former of these methods all the vaso-motor fibres were destroyed excepting those which directly supply *some* of the blood-vessels of the brain;* while, by the latter, the greater part, though not all, of these nerves were rendered functionless. The secondary rise in pressure following the fall produced by an injection of saponin directly into the heart also ceases to occur after section of the spinal cord in the neck.

The cause of the augmentation in the blood-pressure following the introduction of saponin must therefore be stimulation of the vaso-motor "centre." Whether this stimulation is directly produced by the action of the drug on the "centre," or whether it occurs reflexly from irritation of sensory nerves, will be discussed later.

When a large dose of saponin is injected into the femoral vein, the same palsy of the heart is produced as follows the injection of a much smaller dose into the jugular vein.

No. of Experiment.	Animal.	Time.	Pressure.	REMARKS.
43.	Small Rabbit.	Dec. 24, 1877.		Canula in carotid. Vagi cut.
		1 sec.	67.3 mm.	
		3 "		.05 gram. into femoral vein.
		7 "	48.9 "	
		10 "	23.6 "	
		15 "	Negative.	Animal dead.
44.	Cat.	Dec. 21, 1877		Canula in carotid.
		1 sec.	102.7 mm.	
		5 "		.15 gram. into femoral vein.
		10 "	73.4 "	
		15 "	46.5 "	
		25 "	Negative.	Animal dead.

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* That nerves (vaso-motor?) proceed from cerebral cells to the coats of the blood-vessels I have often observed.

This fall in the pressure, immediately following the injection of a large dose of the glucoside into the femoral vein, must not be confounded with that which succeeds the rise in animals where but small doses were injected. The former is due to paresis, or paralysis of the heart, while the latter, not occurring after section of the cervical pneumogastrics, is due to stimulation of these nerves.

*The action of saponin on arterial pressure when injected into an efferent blood-vessel.**

The injection of the drug into the internal carotid artery towards the brain.

No. of Experiment.	Animal.	Time.	Pressure.	REMARKS.
46.	Albino Rabbit.	Oct. 7, 1877.		Canula in carotid.
		10.22	52.4 mm.	
		10.22.30	52.4 "	
		10.23	52.4 "	
		10.24	52.2 "	
				.025 gram. into int'l carotid.
		.05	54. "	
		.10	56.2 "	
		.15	61. "	
		.20	64.1 "	
		.30	62.5 "	Clonic convulsions.
48.	Dog.	Nov. 7, 1877.		Canula in carotid.
		1 sec.	127.1 mm.	
		5 "		.1 gram. into internal carotid.
		10 "	138.8 "	
		15 "	144. "	
		20 "	149.4 "	
		30 "	157. "	
		120 "	104.6 "	

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* The veins of the portal circulation are included under this head.

From these experiments it will be seen that saponin thus introduced, produces a rise in the arterial pressure, which rise in almost all my experiments was succeeded by a fall. The same effect was observed when the drug was injected into the portal circulation.

No. of Experiment.	Animal.	Time.	Pressure.	REMARKS.
49.	Large Rabbit.	Dec. 6, 1877.		Canula in carotid.
		1 sec.	82.8 mm.	
		5 "		.05 gram. into mesenteric vein.
		10 "	86.5 "	
		13 "	86.8 "	
		15 "	82.1 "	
		17 "	64.9 "	
		30 "	61.6 "	

An injection into a femoral artery is followed by the same effect.

No. of Experiment.	Animal.	Time.	Pressure.	REMARKS.
50.	Dog.	Dec. 13, 1877.		Canula in carotid.
		1 sec.	152.7 mm.	
		5 "		.2 gram. into femoral artery
		10 "	165.3 "	
		20 "	171.2 "	
		30 "	175.9 "	
		60 "	172.8 "	
		120 "	144.1 "	

That the rise in the blood-pressure produced in the above experiments was not due to paralysis of the pneumogastrics was determined by previously cutting these nerves, and then injecting the drug as before, when a rise in the pressure still occurred. The secondary fall in the pressure, however, fails to occur in animals thus prepared.

No. of Experiment.	Animal.	Time.	Pressure.	REMARKS.
51.	Cat.	Nov. 30, 1877.		Canula in carotid. Vagi cut.
		1 sec.	110–113 mm.	
		5 "		.2 gram. into intl. carotid.
		10 "	130–136 "	
		15 "	132–138 "	
		20 "	150–154 "	
		30 "	149–153 "	
		55 "	142–145 "	
		120 "	110–113 "	

If the rise in the blood-pressure fails to occur when the spinal cord is cut in the upper dorsal or cervical regions, it must be due to vaso-motor stimulation.

No. of Experiment.	Animal.	Time.	Pressure.	REMARKS.
52.	Dog.	Dec. 20, 1877.		Section of medulla at calamus. Canula in carotid. Vagi cut.
		1 sec.	115–125 mm.	
				.1 gram. into femoral artery.
		10 "	115–125 "	
		15 "	115–125 "	
		30 "	115–125 "	
		60 "		.1 gram. into femoral artery.
		70 "	115–125 "	
		80 "	115–125 "	
		120 "	115–125 "	

No. of Experiment.	Animal.	Time.	Pressure.	REMARKS.
53.	Dog.	Dec. 21, 1877.		Spinal cord cut at twelfth dorsal vertebra. Canula in carotid.
		1 sec.	119–121 mm.	
		5 "	119–121 "	.1 gram. into a mesenteric vein.
		10 "	119–121 "	
		15 "	119–121 "	
		30 "	119–121 "	
		60 "	119–121 "	
		70 "		.1 gram. into femoral artery.
		80 "	119–121 "	
		90 "	119–121 "	
		2 min.	119–121 "	
		2.30		.1 gram. into artery of thorax.
		.35	125–130 "	
		.40	140–145 "	
		.45	162–164 "	Tetanus.
		3.		Section at calamus; tetanus immediately ceased.
		3.20	98.2 mm.	
		.30		.1 gram. into artery of thorax.
		.34	98.2 "	
		.50	98.2 "	
		4.	98.2 "	
		5.	98.2 "	
		7.	98.2 "	

From these and other similar experiments, I arrived at the conclusion: that, the increase in the arterial pressure from an injection of saponin into an efferent blood-vessel does not occur if the spinal cord is cut high up, and consequently it must be due to stimulation of the vaso-motor "centre." This "centre" may be stimulated either directly by the drug itself or reflexly from the action of the drug on the peripheral ends of the afferent nerve fibres. Were it due to the latter it is very apparent that if the connection of the sensory nerves of one posterior extremity with the spinal cord and brain is destroyed, and an injection made into an artery of that limb, the increased blood-pressure would not occur. If in the same animal, however, the drug was introduced into an artery of another extremity where the nerves were uncut, the rise in the pressure would still occur, provided it were due to a reflex stimulation of the vaso-motor "centre."

No. of Experiment.	Animal.	Time.	Pressure.	REMARKS.
54.	Dog.	Dec. 6, 1877.		Canula in right femoral artery. Sciatic and anterior crural nerves cut on right side.
		1 sec.	107-5 mm.	
		3 "		.1 gram. into right femoral artery.
		7 "	107-5 "	
		14 "	104.1 112.4	
		30 "	107-5 mm.	
		40 "	107-5 "	
		60 "	107-5 "	
		120 "	107-5 "	
		4 min.		.1 gram. into right femoral artery.
		4-10 sec.	107-4 "	
		-20 "	107-6 "	
		-30 "	107-4 "	
		-40 "	107-5 "	
		-60 "	107-6 "	
		10 min.		.1 gram. into left femoral artery.
		-10 sec.	107-5 "	
		-20 "	111-6 "	
		-40 "	117-5 "	
		-60 "	112-1 "	
		11-40 "	101-4 "	
		23 m.	105-4 "	.1 gram. into left femoral artery.
		-10 "	109-6 "	
		-30 "	107-3 "	
		-50 "	108- "	
		28 m.	106-6 "	.1 gram. into right femoral artery.
		-10 "	106-6 "	
		-20 "	106-6 "	
		-30 "	106-6 "	
		29 m.	106-6 "	

A large number of similar experiments gave results identical with the above.

As was before shown (Experiments 46, 48 and 51,) an injection into the carotid artery towards the brain produces the same augmentation in blood-pressure. It was necessary to determine whether this augmentation was due to the direct effect of the drug on the vaso-motor "centres," or to an effect on fibres of the cerebrum, afferent to, and regulating the action of, this "centre." To do this a complete section of the pons was made and a canula placed in the femoral or carotid artery. If saponin was now injected into the carotid artery no effect on pressure occurred, though the usual result was observed when the drug was injected into a femoral artery.

No. of Experiment.	Animal.	Time.	Pressure.	REMARKS.
55.	Cat.	Dec. 16, 1877.		Section of pons. Canula in carotid.
		1 sec.	92.7 mm.	
		3 "		.1 gram. into carotid artery.
		10 "	92.7 "	
		15 "	92.7 "	
		18 "	92.6 "	
		30 "	92.7 "	
		40 "	92.7 "	
		60 "	92.7 "	
		3 min.	92.7 "	.1 gram. into right femoral artery.
		10 sec.	98.3 "	
		20 "	103.4 "	
		30 "	107.6 "	
		4 min.	104.2 "	
		6 "	87.4 "	
		7 "	92.7 "	
				Cut sciatic and anterior crural nerves on right side.
		10 min.	92.6 "	.1 gram. into right femoral artery.
		10 sec.	92.6 "	
		20 "	92.6 "	
		40 "	92.6 "	
		11 min.	92.6 "	

The irritative effect of a saponin solution on the fibres of the cerebrum, which are afferent to the vaso-motor "centre," far exceeds that which is brought about by applying a strong induction current to the anterior crural nerve. In dogs, where the medulla oblongata and pons were so mutilated that strong irritations of the sciatic nerves failed to produce any change in the blood-pressure, a solution of saponin introduced into the internal carotid artery immediately occasioned a decided rise in the mercury of the manometer.

CONCLUSIONS.

1. *Saponin directly introduced into the jugular vein of a small mammal, or into the heart of a large animal, produces immediate cardiac paresis, and consequently a diminished arterial pressure.*
2. *The same dose of this drug injected into the femoral vein or an efferent blood-vessel produces: first, an augmented, followed by a diminished arterial pressure.*

The augmentation from the latter method, and probably also from the former, is due to the drug acting as an irritant to the afferent nerve fibres, and thereby, reflexly, stimulating the vaso-motor "centre."

The secondary fall in the blood-pressure produced by both these methods of introduction, is due to stimulation of the pneumogastric centres. This is also reflexly produced, because it does not occur if the afferent nerves of the limb into which the drug is, peripherally, introduced, are destroyed.

3. *Saponin is fatal in small doses when injected directly into the heart. It requires much larger doses to produce death when introduced into the femoral vein, and exceedingly large doses are required if the injection is made into an efferent blood-vessel.*

ACTION ON THE PULSE.

Bonneau and Malapert* found that when plants which owed their active properties to the presence of saponin were introduced into the alimentary canal of dogs, an increased frequency of the pulse was produced.

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* *Casper's Vierteljahrs. f. ger. u. œf. Medic.*, B. 2, Hft. 1, p. 101.

Hoppe* says that saponin first excites and then depresses the heart's action.

In frogs and rabbits, according to Köhler,† the pulse is slowed when this glucoside is introduced into the circulation.

Przybyszuoski‡ found saponin to slow the pulse.

The apparatus employed in my experiments on this subject was a Marey tambour, which was attached to the carotid, and made to write on a drum revolving at the rate of 60 cent. per minute.

An injection into the jugular vein of rabbits and cats, produces a marked diminution in the pulse-rate, which at the same time becomes very irregular.

No. of Experiment.	Animal.	Time.	Pulse.	REMARKS.
1.	Albino Rabbit.	Dec. 22, 1877.		Canula in carotid.
		3.14	96–200pm	
		.15		.05 gram. into jugular vein. Immediate cessation of heart beats, which soon recommenced.
		.17	148 "	
		.18	18 "	
		.20	0 "	Heart ceased to beat, but is still irritable.
3.	Large Cat.	Dec. 3, 1877.		Canula in carotid.
			123–129pm	
				.07 gram. into jugular vein.
			120 p.m.	
			114 "	
			107 "	
			118 "	
			120 "	
			123 "	

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* *D. Nervenwirkung d. Arzneimittel*, II. IV., p. 137.

† *Arch. f. exp. Path.*, B. 1., p. 138-162.

‡ *Arch. f. exp. Path.*, B. 5, p. 137.

That this diminished frequency of the heart's action is not due to an effect on the nervous centres or pneumogastric nerves was shown in experiments where I destroyed these and then injected the drug.

No. of Experiment.	Animal.	Time.	Pulse.	REMARKS.
4.	Rabbit.	Nov. 19, 1877.		Canula in carotid. Vagi cut.
		1 sec.	6 in 1 sec.	
				.02 gram. into jugular vein.
		10 "	4½ " "	
		14 "	4½ " "	
		17 "	4¼ " "	
		24 "	4 " "	
7.	Cat.	Nov. 24, 1877.		Canula in carotid. Vagi cut.
		1 sec.	12 in 2 sec.	
				.015 gram. into jugular vein.
		10 "	12 " "	
		16 "	9 " "	
		60 "	10 " "	

The same effect was observed when saponin was applied to, or injected into the heart of frogs, in whom the central-nervous system had been destroyed.*

The diminished frequency of the pulse, under these circumstances, occurring independent of the vagus and brain, must be the result of an intra-cardiac action of the drug. If it be cardiac, there remains still to determine whether it be due to an effect on the heart muscle itself, or on the nerves and ganglia of this organ. Were the cardiac ganglia possessed of automatism, as is claimed by the majority of modern physiologists, the explanation would readily be found in supposing saponin to diminish their activity. This explanation would be greatly assisted, if not confirmed, by the frequent persistence of cardiac irritability after cessation of the heart's beat. But as these ganglia have by no means been *proven* to be the cause of the movements of the heart, it would not be proper to conclude that a drug influences them either as a paretic or as an inhibitant. The only conclusion which can with certainty be drawn, is that the diminished frequency of the pulse following an injection of saponin into the jugular vein (*i. e.*, the heart), is due to a paretic action on the intrinsic structures of this muscle whose function it is to cause the heart to continue to beat after it is removed from the body of an animal.

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* See, Local action on the heart.

When the drug is introduced into an afferent blood-vessel at some distance from the heart, or into an efferent blood-vessel, an increased followed by a decreased frequency of the pulse occurs.

No. of Experiment.	Animal.	Time.	Pulse.	REMARKS.
11.	Rabbit.	Feb. 12, 1878.		
			29 in 10 s.	
			29 " "	
			28 " "	
				.02 gram. into femoral vein
			33 " "	
			35 " "	
			25 " "	
			22 " "	
16.	Dog.	Dec. 12, 1877.		Canula in femoral artery.
		1 sec.	5 in 2 sec.	
				.125 gram. into femoral vein.
		12 "	6 " "	
		20 "	6¼ " "	
		40 "	6¼ " "	
		5 min.	4¼ " "	

An increased pulse-frequency, following the introduction of a drug at some distance from the heart, may be caused: first, by a cessation or diminution of the cardio-inhibitory action of the pneumogastrics; second, by a stimulation of any or all the nerves concerned in accelerating the heart's beat;* third, by a stimulation of the heart itself.

First, is the accelerated heart's action due to a diminished cardio-inhibitory action of the pneumogastrics?

It is well known that irritation of the neck pneumogastrics either arrests, or diminishes the number of heart beats, and if these nerves are paralyzed the heart beats are more frequent. To determine whether saponin paralyzes these nerves, two series of experiments had to be made: first, to determine whether in a poisoned animal irritation of the neck vagi still produces cardiac inhibition; second, whether after section of the pneumogastrics the glucoside still causes the heart's beat to increase in frequency.

Is the cardio-inhibitory-action on the heart lost?

Experiment 19. Cat. Vagi cut and electrodes from a DuBois Reymond induction apparatus (Leclanche cell) applied to them. Pulse 40-44 in 10 seconds. Irritation of vagi: Pulse ceases. Cessation of irritation, 48, 46, 42 in 10 seconds. Injected .075 gram. of saponin into femoral vein. Pulse, 50-54 in 10 seconds. Irritation of vagi: Pulse, 58 in 10 seconds. Cessation of irritation. Pulse, 59, 52, 49. Irritation of vagi: Pulse, 62 in 10 seconds.

Experiment 21. Rabbit. Prepared as above. Pulse, 28 in 10 seconds. Injected .15 gram. saponin into femoral artery. Pulse, 31 in 10 seconds. Irritation of vagi: Pulse, 35, 36, 38 in 10 seconds, Cessation of irritation. Pulse, 25, 28, 33.

Experiment 22. Dog. Prepared as in other experiments. Injected .25 gram. into the carotid artery. Pulse $11\frac{1}{2}$ to 12 in 10 seconds. Irritation of vagi: Pulse, $15\frac{3}{4}$, $17\frac{3}{4}$, 18 in 10 seconds. Cessation of irritation. Pulse, 13, 15 in 10 seconds. Irritation of vagi: Pulse, 18, $19\frac{1}{8}$ in 10 seconds. Cessation of irritation. Pulse, 15, 14 in 10 seconds.

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* It has been contended that the blood-pressure mechanically influences the frequency of the pulse, but recent experiments have shown that this influence is only exerted indirectly through the heart-nerves.

From the above experiments it will be seen that, after an injection of saponin, irritation of the neck-pneumogastrics not only fails to produce heart-arrest, but on the contrary, causes cardiac excitation.* This shows that these nerves contained heart-accelerating as well as heart-inhibiting fibres. These experiments, however, do not necessarily prove that the inhibitory nerves are paralyzed, as it is possible that they remain unaffected while the excitability of the accelerating heart fibres of the pneumogastrics was increased by saponin.

As no other direct cardiac inhibitor is known to exist, I in a number of experiments cut the pneumogastrics and then gave the poison as before. If now the frequency of the pulse still increases, this result cannot be due to paralysis of these nerves.

No. of Experiment.	Animal.	Time.	Pulse.	REMARKS.
23.	Rabbit.	Jan. 2, 1878.		Cut vagi in the neck.
			17 in 10 s	
		1.58		.02 gram. into femoral vein.
		.15	21 " "	
		1.59	20 " "	
		.30	18 " "	
		2.00	15 " "	
		2.01	15 " "	
25.	Dog.	Dec. 8, 1878.		Pneumogastric, sympathetic and recurrent nerves cut in the neck.
			$3\frac{1}{3}$ in 2 s.	
				.1 gram. into femoral vein.
		15 sec.	6 "	
		25 "	6 "	
		30 "	6 "	
		35 "	$3\frac{1}{8}$ "	
		60 "	$2\frac{7}{8}$ "	

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* That the pneumogastrics contain accelerator nerves of the heart has frequently been shown by Moleschott, Schiff, Schmiedeberg, and others. After an injection of saponin, irritation of the vagi also produces a rise instead of a fall in the blood-pressure.

From a large number of experiments, similar to the above, I was led to the conclusion that neither the acceleration nor the diminution of the heart's beat was produced by any action on the pneumogastrics, neck sympathetic or recurrent laryngeal * nerves.

The only other nerves which have been asserted by physiologists to contain cardiac accelerators are the spinal branches of the ganglion stellatum of the sympathetic nerve. In order to determine whether or not the excitation of these nerves through saponin caused the increased frequency of the pulse, it was necessary to cut the spinal cord above their origin and then examine the effects of the poison.

No. of Experiment.	Animal.	Time.	Pulse.	REMARKS.
28.	Dog.	Dec. 14, 1877.		Spinal cord cut at fifth cervical vertebra. Vagi cut.
			6–6½ in 2 s.	
				.1 gram. into femoral vein.
		10 sec.	6¼ "	
		20 "	6½ "	
		30 "	6 "	
		40 "	6 "	
		50 "	6 "	
		60 "	6 "	
		2 min.	6 "	
		3 "	6 "	
		5 "	32 in 10 s.	
29.	Cat.	Dec. 6, 1877.		Cord cut at fifth cervical vertebra.
			23 in 10 s.	
				.1 gram. into femoral vein.
		10 sec.	23 "	
		20 "	24 "	
		30 "	23 "	
		40 "	22 "	
		2 min.	22 "	
		5 "	22 "	

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* That the recurrent laryngeal nerves contain heart accelerators has been abundantly demonstrated by the experiments of Schiff (*Altes u. Neues ueb. Herznerven*, etc.).

The acceleration of the heart's beat not occurring after section of the spinal cord above the origin of the spinal cardiac accelerators, it very likely is due to stimulation of these fibres or their centres. As the secondarily produced diminished frequency of the pulse also fails to be present after this operation, the conclusion must be drawn that this is due to a secondary paretic effect on these nerves or their centres. These centres must exist in the central nervous system below the pons, as it was found that, after complete section of the pons, an injection of saponin into the femoral vein still produces its characteristic effect on the pulse.

No. of Experiment.	Animal.	Time.	Pulse.	REMARKS.
30.	Dog.	Dec. 29, 1877.		Section of pons and vagi.
			20 in 10 s.	
				.1 gram. into femoral vein.
			35 " "	
			37 " "	
			38 " "	
			38 " "	
			38 " "	
		5 min.	16 " "	

The primary excitation and the secondary paresis of these nerves must be produced reflexly through stimulation of afferent nerves; for if the afferent nerves of a limb are cut, and saponin is injected into its artery, they fail to occur, though the rest of the nervous system be normal. This is an interesting physiological phenomenon, the possibility of which has never before been demonstrated.

No. of Experiment.	Animal.	Time.	Pulse.	REMARKS.
32.	Dog.	Dec. 18, 1877.		Canula in femoral artery. Sciatic and anterior crural nerves cut on the right side.
			6½–6¾ in 2s.	
				.1 gram. into right femoral artery.
		7 sec.	6¾ "	
		14 "	6¾ "	
		40 "	6¾ "	
		60 "	6½ "	
		4 min.	6¾ "	
				.1 gram. into right femoral artery.
		20 "	6¾ "	
		40 "	6½ "	
		5 min.		.1 gram. into left femoral artery. On this side the nerves were intact.
		10 "	7 "	
		20 "	8¾ "	
		30 "	8½ "	
		40 "	8 "	
		50 '	8 "	
		6 min.	2⅓ in 5s.	
		10 "	5 "	
		40 "	2⅓ "	
		11 min.	5½ "	
		40 "	6 "	
		23 min.	5¾ "	.1 gram. into left femoral artery.
		10 "	6¼ "	
		40 "	7¾ "	
		50 min.	3¾ "	

CONCLUSIONS.

1. *Saponin directly introduced into the heart diminishes the frequency of the pulse through its paretic effect on the excito-motor mechanisms of this muscle.*

2. *Introduced into a vein at some distance from the heart, or into an efferent blood-vessel, it reflexly excites the accelerators of the heart which exist in the cervical spinal cord. The excitation is followed by paresis of these nerves. Both the paresis and the excitation are due to the action of the drug on the afferent nerve fibres.*

ACTION ON THE RESPIRATORY SYSTEM.

The local action of saponin on the mucous membrane of the air passages was treated of on one of the earlier pages of this work. Introduced anywhere into a living animal it produces an increased mucous discharge from the bronchial tubes. How it produces such an increased secretion I shall here endeavor to explain.

A well known fact in the physiology of secretion is, that an increased elimination from a secreting surface must be due to an increased blood-supply to its secreting apparatus. So it is with the lungs: saponin introduced into the stomach, into the blood-vessels, or endermically, invariably produces hyperæmia of the lungs. This, it may be contended, is only a collecting of the blood in the radicles of the pulmonary vein. Careful examination has, however, convinced me that not only do the pulmonary vessels contain more blood than normally, under these circumstances, but the bronchial vessels are also surcharged with this fluid. Even going aside from these observations, anatomists have shown that communications exist in the lungs between the bronchial and pulmonary vessels. So that if one set of vessels be surcharged with blood the other set must be similarly affected. *The increase in the bronchial discharge must therefore be due to the increased vascularity of the lungs* which follows the introduction of this drug.

To what is the increased vascularity due?

Hyperæmia of the lungs may be caused by contraction of the left auriculo-ventricular valve impeding the outflow of the blood from the left auricle, and also by insufficience of this valve with systolic regurgitation from ventricle to auricle, and as a consequence the left auricle and pulmonary vein can empty their contents only with great difficulty. Under these circumstances, according to Niemeyer,* "Bronchial catarrh is not only one of the most constant, but one of the most physiological and inevitable symptoms." As, however, saponin does not produce "heart murmurs" of any kind, these causes may be excluded.

The only other known cause to account for the hyperæmia of the lungs is, paralysis of the vaso-motor nerves of these organs. On examining the various monographs on the vaso-motor nerves, I have been struck with the utter lack of allusion to the vascular nerves of the lungs, so that on commencing the experiments on the respiratory action of saponin I was compelled to precede them by other experiments to determine which were these nerves.

The vascular nerves of the lungs certainly do not arise from the spinal cord, as section of this at any point below the medulla oblongata is followed by anæmia instead of hyperæmia of the lungs. The only other nerves which directly supply these organs are the pneumogastrics, section of which has been known since the beginning of the present century to produce an increased vascularization of the lungs. So that it seems very probable that vaso-motor nerves for the lungs exist in these nerves, an inference which my experiments on saponin have confirmed.

When the hyperæmia has been produced by the drug, irritation of the cervical pneumogastric trunks causes the hyperæmia to disappear immediately, to reappear again on removing the irritation. This experiment was repeatedly made with unvarying results. This effect of the pneumogastrics on the vascular supply of the lungs is not due to inhibition of the heart; as in saponized animals irritation can no longer produce inhibition. *The cause of the increased vascularity of the lungs is very probably to be found in a paresis of the respiratory vascular nerves.*

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* *Practical Medicine*, Am. Ed., V. 1, p. 60.

Saponin also affects the respiratory movements, making them irregular in rhythm and usually more frequent.

Experiment on a rabbit. Respirations, 60 per min.

11.53. Injected .01 gram. into femoral vein.
11.56½. Respirations, 67 per minute.
11.59. " 64 "
12.02½. " 64 "
12.04. " 66 "
12.12. " 64 "
12.24. " 66 "

Experiment on a dog. Respirations, 9 per minute.

9.56. .1 gram. of saponin into femoral vein.
9.56½. Respirations, 12 per minute.
9.58. " 18 "

Though in these and other similar experiments a decided increase in the number of respiratory movements occurred, in quite a number of other experiments the opposite result was obtained. When, however, the cervical pneumogastrics were previously cut, an injection of saponin almost always produced a diminution in the number of respiratory movements, showing that the opposite effect on animals in whom these nerves have not been cut, is very probably produced by irritation of the afferent pneumogastric fibres in the lungs.

The usual effect in non-poisoned animals of irritation of the peripheral extremities of the cut cervical pneumogastrics is to decrease the number of respiratory movements; but after an injection of saponin irritation of these nerves increases the number of these movements.

Experiment on a cat. Vagi cut. Respirations, 12 per minute. Irritation of vagi: Respirations, 10 per minute.

Injected .1 gram. saponin into the femoral vein. 3 minutes after the injection: respirations 9 per minute. Irritation of the peripheral ends of the cut vagi increase their number to 12 per minute.

ACTION ON THE SECRETORY ORGANS.

A solution of the drug introduced into the general circulation apparently does not affect the secretions from the salivary, biliary, and renal glands. Through the aid of fistulas it was determined that no change in the amount secreted took place from any of these glands. With the gastric juice the result was different, its secretion was increased.

The local irritant effects of saponin on the stomach in producing vomiting, etc., have already been noticed. Vomiting is, however, also produced when this drug is introduced into the general circulation. This symptom may be produced by an action on the vomiting "centre" in the medulla oblongata; it may be reflex: or it may be of gastric origin. If it be the latter, saponin must be eliminated by the stomach. I have found in a number of experiments that while the normal gastric juice contains no substance which reacts like our glucoside, yet 15–30 minutes after the injection of a solution of the latter into a vein, or several hours after its introduction endermically, saponin can be detected in the secretion of the stomach. In the matters vomited by these animals traces of it were also found. It appears from this that *the vomiting produced by this drug is always of gastric origin.*

The peristaltic movements of the intestines are much increased by saponin. This effect may be due to paralysis of the inhibitory fibres (splanchnici) of these structures, or to a stimulation of their excito-motor nerves (pneumogastrics ?).

Pflüger* found that irritation of the splanchnic nerves produced peristaltic inhibition. If the increased peristalsis from saponin was due to paralysis of these nerves, their irritation would no longer inhibit these movements. But as their irritation still inhibited the peristaltic movements, *the increased peristalsis from saponin must have been due to stimulation of the excito-motor nerves of these viscera.*

ABSORPTION AND ELIMINATION.

That saponin is absorbed is beyond doubt, as its general effects are manifest even when it is introduced into the stomach or endermically. That it is eliminated as saponin by the stomach and liver was proven by numerous experiments. The secretions from these organs invariably reacting for the glucoside after its introduction into the general circulation, but do not affect the re-agents normally.

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* *Ueb. Hemmungsnervensystem f. d. peristal. Beweg. d. Gedarme.* Berlin, 1857.

Whether the drug is eliminated by the urine could not be determined, as the presence of the phosphates and sulphates too greatly interfered with the tests.

Is saponin destroyed by the liver? This was a very difficult question to answer, but after making a large number of experiments, I am now able to say that the drug is not destroyed, but is eliminated by the liver.

The circumstance that saponin is poisonous in smaller doses when introduced into the jugular vein, than when it is injected into the portal circulation, does not aid us here, as by the former method we have the direct effect on the heart which so rapidly kills the animals experimented upon. Saponin is also less poisonous if injected into the femoral artery, than if it was introduced into the portal circulation.*

In the frog experiments more positive results were obtained; but even these, as will be seen later, only demonstrate that the liver eliminates the poison. The drug used was poisonous to medium-sized frogs in doses of .0035 gram., while doses of .0023 gram. failed to produce fatal results.

When, however, the livers of these animals were removed, the last mentioned dose invariably proved fatal.

Aug. 30, 1877. Experiment on a medium-sized frog. Extirpated the liver. 1.23. Injected .0023 gram. of saponin into the lymph sac of the back. This dose produced no symptoms previous to the removal of liver. 2.00. Voluntary and reflex movements have ceased. 2.10. The circulation in the capillaries of the web of the foot has ceased. Muscles and nerves are still irritable.

Aug. 25, 1877. Experiment on a medium-sized frog (*R. esculenta*). 10.30 A. M. Injected .0023 gram. into lymph sac of back. No symptoms were produced.

Aug. 31, 1877. Extirpated the liver of a large female frog (*R. temporaria*) at 2.48 P. M.

Sept. 1, 11 A. M. Animal is as well as ever and very lively. 11.01. .0023 gram. saponin into back.

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* Nicotine gives somewhat similar results.

11.45. The animal moves with great difficulty.

4. P. M. Tetanus, no voluntary movements.

4.35. Reflex movements have ceased.

Though large numbers of similar experiments were made, the above will suffice to show that the presence or absence of the liver greatly influences the dose of saponin necessary to kill such an animal. This however does not prove its destruction by the liver. If the poison be eliminated by this organ, as has been proven, it is very apparent that its removal will cause a greater amount of the poison to remain in the system. As a consequence the effects of saponin are greater in liverless than in normal animals.

To determine beyond a doubt, whether the drug is or is not destroyed by the liver, it was necessary to tie the gall-ducts of a frog, and then observe the effects of a dose of the poison which under ordinary circumstances would not kill the animal. If this be done the liver is still able to destroy the drug though it is unable to eliminate it.

Sept. 3, 1877. Experiment on a large frog (*R. esculenta*).

9.45 A. M. Ligated gall-bladder and bile-ducts.

11 A. M. No symptoms.

11.10 A. M. Injected .0023 gram. saponin into back.

12 M. Voluntary and reflex movements are almost abolished.

1.27 P. M. The animal is dead.

Sept. 4, 1877. Experiment on a medium sized frog (*R. esculenta*).

9.08 A. M. Injected .0023 gram. into back.

11.23 A. M. No symptoms.

1 P. M. No symptoms.

4 P. M. No symptoms have occurred.

Sept. 5, 1877, 10.31 A. M. Ligated gall-ducts.

11.17 A. M. Injected .0023 gram. into back.

By 1.30 P. M. the frog was dead.

From these experiments it appears that saponin is not destroyed but is eliminated by the liver. The fact, previously mentioned, that when a mammal dies from saponin-poisoning the presence of the poison can be detected in the bile, goes to confirm this. To still further test the correctness of this result I rendered the liver hyperæmic, after the beautiful method of Schiff, by tying the superior vena cava. If saponin is removed by the liver a greater quantity will be eliminated when this vein is tied, forcing all the blood from the abdomen and lower extremities through this organ. In a number of frogs I made this operation, and then injected poisonous doses of the drug without producing fatal results.

Sept. 3, 1877, 10.30 A. M. Ligated the superior vena cava of a medium-sized frog.

12.27 P. M. Injected .004 gram into back.
12.47 P. M. No symptoms.
1.00 “ “ “
2.02 “ “ “
2.30 “ “ “
3.00 “ “ “
3.15 “ “ “
3.30 “ “ “

Sept. 4, 10.15 A. M. As yesterday, with the exception of an inclination to keep quiet.

From the above given facts the conclusion must be drawn that *saponin is eliminated and not destroyed by the liver.*

EXPERIMENTS ON THE CHEMISTRY OF VARIOUS PLANTS CONTAINING SAPONIN.

Saponin, as was stated on one of the early pages of this work, is contained in a large number of plants. My investigations were made on the *Saponaria officinalis*, *Quillaya saponaria*, and *Polygala senega.* All these contain, besides saponin, kino-tannic acid. This can be extracted from the filtered watery extract of the drugs, by means of ether, and also by maceration with ether and then filtering. By either of these methods quite an appreciable quantity of saponin was extracted with the acid, as was determined by the physiological, as well as by the chemical tests. For these tests the ethereal solution was evaporated over a sand bath and the residue was dissolved in water. This solution was now treated with boiling alcohol, which precipitated the glucoside. A watery solution of this precipitate reacted in every way for saponin;—a precipitate was given with soluble lead and barium salts;

blood-corpuscles were dissolved by it; and injected into the jugular vein of a rabbit, it produced cardiac paralysis.

After ether has extracted all possible from the drugs investigated, water is allowed to percolate through the residue, when an acid filtrate is obtained. The acid of this filtrate did not react with iron salts, and consequently was neither kino- nor gallo-tannic acid. On evaporating this filtrate to dryness and treating it repeatedly with concentrated ether the acidity gradually disappeared, and only a neutral saponin in a pure state remained. More especially were my investigations directed to the root of the *Polygala senega*, from which Quevenne obtained an acid having the properties of saponin, which was called by him acid saponin or polygalic acid.

Saponin obtained from senega by precipitation from the extract by boiling alcohol is, as Quevenne says, of an acid reaction; but this saponin, after concentrated ether has been allowed to percolate through it for a long time, becomes neutral, though it still presents all the characteristics of the glucoside, showing that the acidity is due to the presence of a foreign acid and not to saponin.

Saponin in combination with sulphuric acid.—When to a solution of the glucoside dilute sulphuric acid is added and the resulting liquid is allowed to concentrate, after a time beautiful acicular prisms are found to shoot out at the edge of the liquid.* These crystals when burned leave but a faint trace of ash, showing that they are crystals of an organic compound. They are freely soluble in water; and when a drop of this solution is added to several drops of blood under the microscope, the corpuscles immediately disappear. Injected into a lymph sac of a frog it produces all the symptoms of saponin poisoning.

Saponin is the only glucoside known to form compounds with an acid and at the same time retain its peculiar properties.

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* I have several times seen very small rhombic crystals in a solution of neutral saponin.

ART. II.—GUNSHOT WOUND OF NECK FOLLOWED BY PARALYSIS OF CERVICAL SYMPATHETIC. INSANITY.

By H. M. BANNISTER, M. D., CHICAGO.

OBSERVATIONS of simple traumatic lesions of the cervical sympathetic are not such rarities as formerly, and one presenting merely the classic phenomena would not, perhaps, be of sufficient interest to justify its publication. The following case, however, is peculiar in that it exhibits a consequence of these injuries that has not, to my knowledge, been heretofore reported, and, imperfect as the record is in many respects, I do not hesitate to offer it to the profession.

Peter Kelly, aged 43, policeman, while in the performance of his duty, on the afternoon of Oct. 30th, 1874, received a pistol bullet wound in the neck. The ball, which was a small one, entered on the left side of the neck close to the anterior margin of the sterno-cleido mastoid muscle, between two and three inches above its lower insertion, and after passing backwards and a little downwards was lost in the tissues and never found. About six hours after the reception of the injury he was seen by Dr. L. H. Montgomery; was suffering considerably from shock, but rallied well, and under very simple surgical dressings, with occasional anodynes, the wound was healed up within a month and the man returned to duty. During this time Dr. Montgomery noticed a decided flushing of the left side of his face, but does not recollect or have notes of any special phenomena, such as alterations of the pupil, etc.

After the healing of the wound the redness of the left side of the face was still noticeable and at times it appeared almost purplish in spots. His wife reports also that now and then that side of the face would change so in appearance as to almost suggest a paralysis. There was also for a short time a peculiar appearance of the eyes, the right being the most prominent, probably from enophthalmus of the left. The general health became impaired, the appetite was very poor, and about four or five months after the receipt of the wound, there began to be observed a change in his general demeanor and disposition; he grew careless and listless, and yet subject to violent fits of passion upon very slight irritation. He neglected his duty so that about this time or a little later he was discharged from the police force. He began to give evidence, also, of delusions, would sit by himself and mutter about shooting, had an idea that people must tell him their secrets, heard voices talking to him (which, however, he admitted to be hallucinations), and became very irritable and dangerous and sometimes, unprovoked, would attack his wife with whatever weapon was at hand. She noticed at these times that if she screamed loudly he would stop as if aroused to consciousness of what he was doing, and would sullenly sit down. This condition of things continued till the summer of 1877, he showing in the meantime, it is said, occasionally some signs of paralysis, and then he became so unmanageable, under the delusion that he was the Catholic Bishop of Chicago, that his wife had him arrested for her own protection.

In January, 1878 he was brought before the Cook County Court at Chicago to be tried, according to the law of the State, for insanity. Dr. Chas. E. Davis, to whom I am indebted for many of the preceding notes, was foreman of the jury, and carefully examined the patient. He was then listless in manner and complained of pains in shoulders and back and in his hips; his pupils were unequal, the right being the largest, and the left side of his face was redder than the other. No ptosis or other ocular symptom than the pupillary inequality was noted. He claimed to be of sound mind and insisted that he was the victim of a conspiracy on the part of his wife and his brother, but was adjudged insane on the facts above given. He succeeded, however, in obtaining a rehearing and was this time discharged as sane. The old symptoms continuing he was brought before the court a third time last November, was found insane and committed to the County Insane Asylum at Jefferson; where he is at the present time.

I had heard a brief account of this case, with others, written by Dr. Davis and read to the Chicago Medical Society just after the first trial of Kelly for insanity, but did recognize it as the same when my attention was called to him at the asylum, by the Superintendent, Dr. Spray, when there on a visit last winter. The peculiarity in his appearance that then especially attracted my attention was an inequality in the two sides of his face, noticeable especially when the muscles of expression were called into play, the wrinkles and furrows between them being more prominent on the left side than on the right. There was also some difference in the color; the pupils were apparently equal, but Dr. Hagenbach, the assistant physician, stated to me that occasionally there was a marked difference. I could not then well examine the case, but on a recent visit, May 17th, the following points were noted:

Patient of medium size, light complexion, fairly nourished, and apparently in good physical health, and with all his bodily functions apparently in good order. It was utterly impossible to obtain from him any subjective symptoms, as he declared himself absolutely well in every respect. His insanity consisted apparently in delusions of grandeur together with ideas of persecution, he was suspicious and a little inclined to bluster, and it was not practicable to make a thorough physical examination for this reason. I could not put a thermometer into the external auditory meatus, nor could I trust it in his mouth; I was therefore compelled to satisfy myself with an observation with a surface thermometer, placed on the side of the face just forward of the ears. This could be relied on only for comparative results, as it was one of the elastic bulb thermometers which are affected by even slight pressure. But with this applied for equal times on both sides of the head, a difference of nearly a degree centigrade in favor of the left side was noted. The left side of the face and especially the pinna of the ear were redder than the corresponding parts on the right side. Since I had seen him before he had let his beard grow, covering a large part of his cheeks and the lower face, but there was still perceptible a difference in the folds or wrinkles when he talked or smiled. The pupils were apparently equal and reacted well to light, at the time of observation, and there was no ptosis. Contrary to my expectation he allowed an ophthalmoscopic examination to be made; having undergone a similar one before he was not suspicious of it. It revealed nothing apparently abnormal; it was made under disadvantages, however, and was not altogether satisfactory. There was no tenderness on pressure in the cervical region,—at least he would admit of none. The heart sounds, as tested by Dr. Hagenbach, were pure; the pulse (taken sitting) was 80, full and compressible.

The scar of the wound was just barely visible, and by careful measurements by Dr. Spray, it was just on the anterior border of the left sterno-mastoid, and two and a half inches above the sterno-clavicular insertion. The bullet had probably cut some of the fibres of this muscle. It must have come very near the common carotid, and it is easy to see how it, in the course it appears to have taken, might have injured some of the fibres of the sympathetic, probably between the middle and inferior cervical ganglia.

On account of the phase of his insanity which has been mentioned, the patient was not a good subject for experiments, and it did not seem advisable to try the effect of drugs or of the reflexes on the condition of his pupil. I therefore asked the assistant physician, Dr. Hagenbach, to carefully observe it at different hours of the day for several days, which he very kindly did for me. The general results he states as follows: "In the ward, where the light is not strong, the right pupil usually a little the larger and more vacillating in size than the left. The difference is not very marked, but can be demonstrated, I think. When exposed to the direct rays from a lamp, both pupils contract equally well, and the vacillation is not noticed."

Other points in regard to which I asked him to observe, were the condition of the secretions, in respect to which nothing notable was remarked, except, perhaps, a greater dryness of the right side of the scalp, and the comparison of the two sides of the face, which I had not made to my own satisfaction. I had thought that there was a very slight hypertrophy of the left side, but Dr. Hagenbach held rather that the difference in the appearance of the two sides was due to a slight paralysis of the right, which made the muscles of the left side more prominent in their action. It is very likely, also, if this is so, that there is a little atrophy on the right from the less use, and that inequality of the two sides actually exists. It is not enough however to be easily ascertained by measurements. Dr. Hagenbach writes me that he had considerable difficulty in making any close examinations, owing to the patient's suspicions.

This case seems to me to be as nearly one of uncomplicated injury to the cervical sympathetic as any I have seen recorded. Instances of this kind are very infrequently met with, since wounds that could involve the sympathetic in this region are almost certain to implicate other important organs, such as the carotids, which are in very close proximity. Cases of disturbance in the functions of the sympathetic from injuries to the spinal cord or brachial plexus, are comparatively common; but I have found the records of only five or six in which the phenomena of an independent lesion were observed. Such are those reported by Mitchell,* Bernhardt,† Kämpf,‡ Flensberg,§ perhaps one of Seeligmüller's,‖ and those reported by Jewell;¶ all but the last of which are referred to by Eulenburg and Guttmann, in their essay on the Physiology and Pathology of the Sympathetic System of Nerves, published in the *Journal of Mental Science* for July and October, 1878, and January, 1879. In the case I have related, there is no history of any symptoms indicating the lesion of other important nerves than the sympathetic, while most of the evidences of its involvement that have been observed in other cases were present. Thus we have reddening and increased temperature of the left side of the face, and enophthalmus and myosis of the left eye. Other symptoms, such as ptosis, may have existed at one time, but the history is defective in many respects, and I have been careful to sift the testimony of unprofessional witnesses, and only give what appear to be indisputable facts.

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* Injuries to Nerves.

† *Berliner klin. Wochensch.*, 1872, Nos. 47 and 48.

‡ Quoted by Eulenburg and Guttmann.

§ Quoted by Eulenburg and Guttmann.

‖ *Berliner klin. Wochensch.*, 1872, No. 8.

¶ This journal, Jan., 1874.

The slight inequality of the two sides of the face, and the partial paralysis of the right side, are of interest. Inequality alone has been observed in other cases, such as those of Seeligmüller and Bernhardt; in these it was the side of the lesion that was emaciated. This is said to be the rule in the second stage of paralysis of the cervical sympathetic by Nicati,* who also says that the primary increase of temperature gives place to a decrease. This last was not observed by either Bernhardt or Seeligmüller, nor by myself in Kelly's case. In all the temperature was highest on the side of the paralyzed sympathetic. In Kelly, also, the disparity in the two sides of the face was in favor of the left—the side of the injury. The paralysis of the right half of the face, whatever there is of it, I can only suppose to be a remote consequence of the wound; perhaps due to secondary intra-cranial disorder.

The main interest in this case is, however, its connection with the mental disorder now manifested by Kelly. There is, so far as can be learned, no history of any special neurotic tendency, no heredity of insanity, and no previous symptoms indicating mental disorder existing anterior to the reception of the wound. His wife states that he was an entirely different man before the injury; and that a change was remarked very soon after, though long before insanity was suspected. There was, in fact, no known cause for the mental derangement other than the wound; and it appears to me a sequence that may not altogether unreasonably be considered a result. It is true that the majority of writers on the pathology of the sympathetic have not included insanity among the diseases due to disturbance of its function; yet if we are to consider the views of one of the most recent works† on mental disorders as well founded, this plays a most important part in their production. The special form of Kelly's insanity is much such as might be expected with the acceptance of Schüle's views, a kind of grand delirium, with ideas of persecution and suspicion. Although he did not present the physical symptoms of general paralysis, at least to any noticeable degree, his manner and general appearance was not unlike that of many paralytics that I have seen. His case, however, is not now, I believe, considered one of general paralysis, and may never develop into it.

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* "La Paralysie du Nerf Sympathetique Cervical." *Inaug. Diss.*, Zurich, 1873.

† Schüle: *Handbuch der Geisteskrankheiten.* Leipsic, 1878.

The fact that other cases of paralysis of the cervical sympathetic do not result in insanity, is not conclusive in any way as regards the present one. Uncomplicated traumatic cases are too rare, and non-traumatic cases are too complicated with other morbid processes, as a rule, to permit of any generalizations. It must be remembered that other special causes of insanity, as well as of other ill-understood nervous diseases, such as tetanus, may occur many more times than their peculiar result. In cases of paralysis of the cervical sympathetic, it is not necessarily to be expected that the resulting cerebral hyperæmia will produce mental derangement; the intimate nutrition of the ultimate psychically functioning structures, which may or may not be seriously affected by the hyperæmia, must first suffer. But that it may have this effect, the case above related seems to me to be a clinical evidence of perhaps some value.

ART. III.—SOME EXCEPTIONS TO THE PRESENT MANAGEMENT OF HOSPITALS FOR THE INSANE.

BY CLARK GAPEN, M. D.,
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THE past score of years has been characterized by vast improvement in the methods of management of the insane, in this country, and yet one cannot remain unconscious of an existing dissatisfaction on the parts, both of the lay and the scientific public, with the present status of Insane Hospital management.

Among scientists it takes definite shape in an open hostility to certain methods of management or treatment, or sharp censure of inactivity in certain directions. With the lay public it amounts only to a vague, indefinite feeling that all is not well, expressed in the easy manner in which it is aroused on such matters, the general readiness to believe all sorts of terrible things regarding the treatment of these unfortunate beings, and, especially, in the general reluctance to place a friend in an institution of this character, however urgent the need, much valuable time often being lost in the generally vain endeavor to benefit the afflicted one at home before seeking the care of the hospital.

That these two circles of dissatisfaction exist few will deny. That they could not well exist without cause all will concede. What may these causes be?

Let us first consider, for a moment, the organization of these institutions. They spring from an urgent need, on the part of the community, for some intelligent and efficient means of treating those afflicted with a malady, which, unlike most other maladies, cannot best be treated in the presence of home influences. In addition to a removal from these, the successful management of the cases requires a means combining comfort and facilities for constant observation and treatment, with so much of confinement or restraint as the individual case demands. These ends are best secured in the erection of large hospitals for this especial purpose.

When erected the Hospital is placed in the care of a Board of Trustees composed of citizens of known integrity of character. They in turn select as Superintendent, generally, one who has had experience in the care and treatment of the insane, and to him is committed the organization of the working force of the Hospital and the details of its management. Supposing the building complete and equipped, the Superintendent's duties consist in its supervision, together with that of the premises; its supply; the selection of proper employees; the examination of applications for admission of patients, and the treatment of those received.

The scheme seems good, and indeed, would be so, were it carried out in accordance with the original intention. But precisely in this lies the difficulty.

Reviewing this scheme, the first feature that arrests our attention is the guardianship thrown about the institution and its inmates, in the form of a Board of Trustees. No one, familiar with such, will question the *integrity* of Boards of Trustees of Insane Hospitals, in this country. As a rule they are composed of the very best and most honored and respected citizens of a State. But their relations to the institution under their care are such as to render their Trusteeship almost *nil*, except as it relates to the most general affairs. The Board makes its regular monthly or quarterly visit, as the case may be, and finds the general appearance of things satisfactory. But it can know nothing, in such a visit, of the thousand minor details which make all the difference between success or failure in the management of the institution, or of the elements of discord or harmony that abide there. Passing hastily through the wards, what can they know of the experience of any individual patient? And even did they wish to make any inquiry, of an attendant or other employee, it would be impossible to elicit anything but that most favorable to the management, owing to the fact that they are overawed by an autocracy which, in its power over those under it, has no parallel in this country; an autocracy having the power, practically, if not theoretically, to make and unmake without being amenable.

It will be conceded, of course, that the treatment of the patients confided to the care of the institution is the ultimate and most important fact; and yet it thus becomes impossible for these well-meaning guardians to know anything of consequence about this very thing. It is difficult, at times, for even the officers of such an institution to inform themselves of the exact condition of the wards. How much more difficult then for those who only visit the institution hastily once a month at most! I do not mean to imply that Boards of Trustees are blamable for this imperfect supervision. The fault lies rather with the scheme that requires so little of them. The offices being non-remunerative, the incumbents cannot afford to devote the amount of time and attention necessary to make them familiar with the real condition of things. Furthermore, the idea has become prevalent among even the best of them that frequent visitation and extended inquiry are synonymous with meddling, which idea is not unlikely to be encouraged and fostered by the officer in charge.

I think it necessary to devote but little space to the consideration of the custom of inviting a member of the medical profession of the State to accompany a number of the Board of Trustees in a monthly visit, for the reason that it simply amounts to nothing at all, a report from such an inspector, generally designated by the high-sounding title of "Chairman of the Visiting Committee," averse to the management being an unheard of thing, his peculiar relations and the nature of his appointment being such that a report less than laudatory would preclude the necessity of his ever preparing another. Indeed it is extremely amusing to run through the reports of various institutions, and see what excellent characters are invariably given to each by those whose especial business, as in this case it is, to criticize the management.

With respect then to the system of guardianship, of visitation and inspection, which the scheme implies, we may therefore summarize, that in so far as it relates to general matters, such as the expenditure of special appropriations, the erection of new buildings, the management of the farm, etc., the system works reasonably well, but as regards the central and all-important fact, the protection and treatment of the insane, it falls very far short.

It fosters, by its short-comings, a system of one-man power and concentrates attention, not upon the patient, but upon the farm, the building, the grounds.

Out of this imperfection there grows another and a greater, viz., the autocracy, to which I have before referred, on the part of which springs up an assumption of infallibility before which all religious assumptions of that character pale. A thing absurd to all intelligent minds but capable of working much mischief. I may be accused of using too strong language in this connection, but its full truth may be attested by those who have lived in an institution of this character—and they only. The love of power is so strong in the human breast, and the invitation to autocratic sway so insidious and potent, and withal, in this instance, so catering to self that few men placed in this position, unguarded, have ever been found possessed of sufficient manhood and forethought to resist it. The opportunity is ever present, the consequences rarely immediate, and generally appear so remote that they constitute no bar. Hence we find that in almost every case, sooner or later, the Superintendent of an institution permits himself, and is permitted, to lower himself from the plane of republican manhood to that of petty autocracy. And I do not believe the men are so largely blamable as the system. No man can afford to remain unwatched—not amenable. It is true that there are those of such narrow intellects and sublime forgetfulness of the rights of others, and the amenities of life, that they find in positions of this character their best opportunities of exhibiting their real characters. But as a rule this spirit of autocracy is a gradual growth of which the individual is largely unconscious, and for which he is almost as largely irresponsible.

I have dwelt so long upon these two defects because I believe that in them we may see the sources of one of our centres of disaffection, viz., that existing in the community at large. Why?

In the first place, the public has in some way become conscious that the system of inspection and supervision provided by law does not accomplish anything near its full purpose. That it falls short in the most important particulars. That while the Board of Trustees does well enough the work of general supervision of expenditure, etc., it knows next to nothing of the individual for whom the expense is incurred. He is left entirely subject to and at the mercy of one will, be that petty or great. For him or for the employee there is practically no appeal—appeal is received and treated as insubordination. Now I say that the public knows this, and any one may satisfy himself on this point by asking his intelligent neighbor for his opinion regarding this matter. How he knows it I am not able fully to explain, but that he is correct I can aver. In the second place this same public knows of the existence of an autocracy—of a one-man power in its midst, a thing which the American public instinctively hates. To this score it may be accredited that an institution not infrequently succeeds in building up immediately about it such a hostility and lack of confidence, that in selecting juries for suits brought for unlawful detention unprejudiced jurors cannot be found. This feeling is generally an undertone, and often surprises both the management of the institution and the community itself when occasion calls it forth.

There are those who will say that the present autocratic system is essential to the success of the hospital. That it is not true is proven by the success of English institutions of a similar character, in which in the past twenty years, the power of the Superintendent and the scope of his activities have been largely curtailed, with the result of sending medical men to medical work and cutting them off from general business affairs for which, for want of training and experience, they are generally unfit, and which only a mistaken notion of importance leads them to prefer to strictly scientific labors. Indeed it is to me one of the anomalies that a medical man who has devoted himself to this work, and with the most wonderful avenues open before him, should prefer farm management and general business affairs to investigation in that most interesting field, the human mind and the cause and alleviation of its diseases.

The above-mentioned curtailment of the powers and duties of medical men in English Hospitals, and their consequent turning to medical work proper, has been characterized by a great advance all along the line in English psychological work, both as regards investigation and treatment, and the activity in these directions was never more apparent than now, while we stand idle with the same opportunities before us, and an abundance of material—cases for observation and pathological material for study—but almost entirely unused, for the simple reason that the Superintendent of an institution devoting himself almost entirely to outside labors, the internal affairs of the Hospital fall so largely upon the assistant medical officers as to leave them no time for even a careful study of each individual case, much less any original investigation; their time being divided between housekeeping, superficial study and hasty empirical treatment of the cases, and correspondence. Is it any wonder that our European co-laborers sneer at us and laugh at our pretended discoveries, which have been antedated twenty years by them?

From this state of things springs the disaffection on the part of the scientists. The age being one of active interrogation of nature and the application of the facts thus elicited to the general good, any straggling along the line inevitably arouses the opposition of those heartily engaged in the work. It is humiliating that while the greatest activity prevails, the world over, in this field, we, here in America, who pride ourselves upon our activity and originality, are not only *not* advancing, but find those engaged in the work unspeakably ignorant as to what is going on in this direction in other parts of the world; the present work of institutions, so far as the patients are concerned, consisting, as an observing medical man, a former resident officer in a Hospital for the Insane has well said, "In feeding and grooming;" the best talent and greatest attention being devoted to showy improvements, or other outside affairs, having at best but a remote connection with the welfare of the patient, and which, if at all necessary, could much better be attended to by one who has devoted himself to business instead of science. But the notion obtains among Superintendents that business affairs are more ennobling and complimentary than professional work; that that which is properly the work of a steward is of more importance than the work of the medical man proper. A review of the proceedings of the "American Association of Superintendents of Insane Asylums" will prove the truth of these statements, as their work is more largely of a general business than scientific nature, and relates rather to the building and managing of Hospitals than the treatment of patients or scientific investigation. Turning to the British Association of a similar character, we find it almost entirely engaged in scientific work, and less exclusive, in fact not at all so, as it admits to membership private investigators and assistant officers of Insane Hospitals, while the American Association persistently excludes both. The notion is prevalent that the reason for this exclusive policy on the part of the Association is the fear that it will be shown that Assistants and private investigators are doing much more to advance the science of psychiatry than the Superintendents themselves. This may be true, and very likely is so, but I think that the exclusive policy is more due to the fear that private investigators and subordinate officers, not being interested in affairs of a general business nature, would obtrude scientific discussions upon the Association while it, as at present constituted, is disinclined to give much notice to that sort of thing.

In short, as a body, it is not interested in scientific work, largely, and in fact regards it as beneath the serious notice of those engaged in large business enterprises which, as a rule, though not without numerous and very praiseworthy exceptions, they manage, not as the property of the State, but as personal estates or petty sovereignties, the main purpose of which they seem to regard is to contribute to their comfort, pleasure and social exaltation. Lest I be accused of injustice to many good and useful men I wish to repeat and emphasize the statement that many noteworthy exceptions to this rule exist; but the petty sovereign air, the luxurious apartments and equipages, the indulgences of æsthetic or whimsical tastes at the expense of the State, are all too common to admit of dispute. He who "goes through the forest and sees no firewood" is not more obtuse than he who fails to observe these things in the average Hospital for the Insane.

It is usually exacted of him who presumes to criticize that he offer something better in place of that which he would destroy. I would not, in this instance, with iconoclastic hands, pull down the present system of guardianship, but would rather seek to make it effective by rendering it the imperative duty of every one assuming the office of Trustee to make frequent, irregular, but ofttimes protracted visits to the institution under his care, for which he should be liberally remunerated. By thus devoting more time to the work of inspection and supervision these officers would be enabled to enter more fully into the details of the management, and the institution be thus saved the innumerable blunders and injustices which a one-man power implies. There should be no escape from amenability on the part of the Superintendent through informality or indifference. He should feel that his every act was known and weighed by those more interested in the welfare of the afflicted ones than in the general appearance of things or in him. Every Board of Trustees should also adopt measures by which it may know of the feeling of an institution as a whole toward its superior officer. This is oftentimes the index of a man's usefulness. If he is repellant, tyrannical, or indifferent he is likely to know little of the real condition of things in his institution, below the surface, except by accident. No man can compel the confidence of his subordinates. He must win it. To be successful he must be loved and respected, not dreaded. But the dreaded man may, on the other hand, for a long time repress expression in the most effective manner. It is rare to find a hospital in which any freedom of speech is tolerated. The rule is evasion and a guardedness which savors strongly of servility, secured by a system of government which can only be compared to that of Russia.

There should also be both a business and medical Superintendent, the duty of the former being to attend strictly to the business affairs of the institution under the careful supervision of the Board of Trustees, and that of the latter the medical and scientific work of the Hospital, and nothing else. To many Superintendents this would be depriving them of the sweet morsel which they roll so consolingly under the tongue, but the better class of such would hail it as a great relief, as time would then be afforded them for work, for which they have long and anxiously awaited an opportunity, and for which they wait in vain under the present *régime.*

Then will genuine progress in the field of practical psychology begin in America. Then and not till then will we have careful study of the cases in our Hospitals, and the application of better methods in their treatment.

ART. IV.—NERVOUS EXHAUSTION, OR NEURASTHENIA, IN ITS BODILY AND MENTAL RELATIONS.

ONE OF A COURSE OF LECTURES ON THIS SUBJECT, DELIVERED AT THE CHICAGO MEDICAL COLLEGE, BY J. S. JEWELL, M. D., SESSION OF 1878–9.

LECTURE II.

GENTLEMEN: In my last lecture I gave you a brief account of the principal phenomena or symptoms of *neurasthenia* or nervous exhaustion, to-day I shall try and explain them. You will remember, that the chief symptoms of neurasthenia, are always loss of nerve-power, and usually excessive increase of sensibility, in some one or more of its various forms.

I shall enter into this subject with some particularity, for simple as the philosophy of neurasthenia is, when it is once clearly stated, I am persuaded but few understand it as they ought to do, in order to make their knowledge of practical value, in dictating rational, consistent plans of treatment. My present aim then, is to state as clearly as I can, the actual physical conditions on which the symptoms depend. I should premise, however, that these conditions are not matters of direct physical demonstration, but of inference. However, this should be no real bar to our belief in the reality of these conditions, provided the inferences which lead the way to them are well based, that is, have sound and broad premises, and if they are the outcome of cautious reasonings founded thereon. In the physical science of to-day, there is clearly too little dependence on, because there is so much ignorance of, the nature and conditions of inference. It is almost habitually placed at a disadvantage as compared with direct sense observation and physical demonstration. But this arises out of wrong conceptions as to its nature and value, as well as out of an over-estimate of the dependence which we may legitimately place on the "evidence of the senses." From these remarks you may gather, that I am aware of the real nature of the description I am about to give, and also of the objections which a too realistic scientific spirit might raise against it as to its substantial correctness. Having said so much aside from the main current of my subject, I will go on without farther interruption.

Broadly speaking there are two grand phases to nutrition. They are strictly complementary to each other. They are *waste*, and following in its wake, *repair*. On the one side there is a wearing out with all that this complex process implies; on the other there is a rebuilding or repair, of the damage suffered by the living, and therefore acting tissues which compose the body. In this process, the blood plays a most conspicuous part, for it not only embodies the materials for effecting the needed repair of damage, for which purpose it is conveyed into the midst of the tissues, and irrigates them, but it becomes on the other hand one of the chief means for conveying from the tissues, to the various emunctories of the body, the waste matter which results from their wear and tear.

The blood in its turn depends, for its richness and perfection as a store-house of materials for the repair of the tissues, on two chief circumstances: that is, the efficiency of the digestive system, and of the lungs, on the one hand, and on the other on the perfection of those purifying processes as regards the blood, which pass under the name of excretory functions. And so we might go farther back in a regressive order, to the character of the food taken, the purity of the air breathed, to the causes or conditions and the consequences of disordered action of the excretory organs, etc. But this would lead us at present too far away from the point to which I wish to direct your steadfast attention, viz. : *the condition of the nerve structures in neurasthenia.* By nerve structures I mean nerve cells and fibres, especially the former, for they are the seats of nerve activity *par excellence.*

I suppose it will be admitted by all, that the nervous, like other tissues, when in action, undergo minute or delicate changes, not only as a *consequence* but in some sense as a *cause* of their action. We are fully warranted in assuming that it is not possible for an impulse to be transmitted along a nerve fibre, whether sensory or motor; that it is not possible for a reflex act to occur, however simple; that it is not possible to think, or feel or exercise volition, without the occurrence of some material change, however slight, some destructive molecular change in the fibres and cells which may have been the seats of such action. At its conclusion, the cells and fibres involved are not in the same physical condition as when the action was begun. However powerless our present methods of physical investigation may be to demonstrate it, yet it must be a fact, all the same, that there has been a more or less delicate, diffuse wearing out or substantial loss in the tissue in question. And this substantial loss, or impairment of structure, is not confined to the surface of the cell or fibre, as it is in the wearing out of some portion of an inanimate machine, but is probably diffused all through the interior of the cell or fibre, from circumference to centre and *vice versa.* It is interstitial. Evidently, this process of wear or waste will be slow or fast, small or great according to the intensity and duration of the action which occasions it. If the process is slight in degree no noticeable symptoms are produced. But if it is carried to any considerable extent, one symptom at least is shown: that is *fatigue.* This arises from and therefore indicates, not only a very considerable degree of material waste or wear, but it indicates and is the direct sign of a waste or *loss of power.* This wear or loss of living substance carries infallibly with it a loss of power, and fatigue or a sense of exhaustion, *as a rule*, is the sign of it. It is a plain indication usually, that the process of material wear or waste of the active tissue, and that corresponding exhaustion of power with which the tissue is stored, has gone as far as it can go without endangering to any unhealthy extent the integrity of the tissue itself. Fatigue is the inarticulate remonstrance from the worn, exhausted tissue, that the process of waste ought to stop, and give place, without let or hindrance, to the opposite process of repair and re-invigoration. This is what we call *rest.* The activities of the worn tissue are made to cease, while the process of interstitial repair is permitted to go on, until the substantial loss is made good, until wasted energy is restored, hand in hand with the repair of structure, and then fatigue gives place to that state of feeling, the which when it has come, we say we are *rested*, and we are ready once more for another period of activity; and so on from fatigue to rest, and from rest to fatigue, so long as the conditions of health are or can be observed. A substantial daily equilibrium is attained and maintained between waste and repair. All other things equal, this is health.

This *temporary fatigue* which I have described is not neurasthenia proper, but the only circumstances which prevent us from calling it neurasthenia are these : *degree and persistence or permanency.* If for any cause (and there are many causes), the degree of material waste and exhaustion of power should be so increased that a healthy degree of repair cannot soon or at all occur under the circumstances of a given case, there would be at once a case of neurasthenia, or nervous exhaustion. There would then be persistent or permanent exhaustion or fatigue, for the reasons I have given, and the state of the nerve cells and fibres involved, as regards structure and power, would be as I have described.

Neurasthenia implies, then, simply the carrying of a natural, healthy process, to an unnatural or unhealthy degree. Nerve cells and fibres are not destroyed, but unduly worn and exhausted, and this varies in degree between wide extremes.

In a well-marked case of neurasthenia, the loss of substance from excessive wear and tear is so great, and the exhaustion of power is so complete, that even a prolonged period of rest, under favorable circumstances, may be insufficient to secure a healthy degree of repair, and restore wasted energies, so as to remove a sense of chronic fatigue, and exchange it for an inward sense of rest, of vigor, of a spontaneous tendency to activity.

Such is neurasthenia, pure and simple, considered apart from its remoter conditions or causes. I hope you have followed me closely enough to get clearly in your minds, what is the presumed actual condition of the nerve structures in neurasthenia.

I am aware that I have been stating matters which are quite well known, at least in a certain sense. But it should be remembered that the golden rule, not only of all teaching, but of all learning, is to begin with things we do know, and guided by the clues of similarity and identity, pass to the unknown. But we must start with the known and hence I begin with what is familiar, in order to lead you to what is not so familiar.

So it turns out, that there is a real, pervasive nutritive lesion of nerve structure in neurasthenia. Waste has morbidly preponderated over repair. The nervous system becomes lean. This involves necessarily, loss of power, and undue sensitiveness, or "shakiness" of the nervous system. It is more easily excited than when healthy, and the excitement when once produced, radiates farther, and endures longer, than in health.

This state or physical condition of the nerve structures, differs in degree in various cases, or at different times in the same case, from a very slight exhaustion, to that which is the most profound. It may extend to the whole nervous system, or to only a small part of it, or if to all parts, unequally so to different parts. It may be in part hereditary, or it may be acquired. It may affect chiefly the bodily functions, or it may affect chiefly the mental functions, as in some cases of melancholia, or it may involve both at the same time in varying ways and proportions, as often happens.

But this nutritive lesion just described is not the only, though the most important, condition of neurasthenia. There is one more at least, which I wish to place distinctly before you. It consists in a morbidly fluctuating blood-supply to the affected parts of the nervous system. The nutritive lesion described, seems always to involve an unsteady circulation of blood in the part which has suffered. Especially is the blood-supply likely to *fluctuate rapidly*, from a too free and tumultuous supply for healthy nutrition, down to a partial anæmia. Nutrition in the part in question is therefore irregular and unreliable. Hence nervous action is unsteady and unreliable.

The condition of the walls of the blood-vessels themselves, and of the local vaso-motor mechanisms which control them, in the diseased nervous centres, is much the same as that of the worn nerve elements in the midst of which they are. The local vaso-motor apparatuses become worn and exhausted, and the muscular walls of the vessels in the seat of disease lose their tonus in a measure, and give way before any increase in blood-pressure with abnormal ease, and this is one of the chief conditions of a fluctuating circulation of blood in the diseased nervous centres. When it is known how sensitive are the higher nervous centres to changes in the pressure and quantity of the blood circulating in them, it will not be difficult to understand how rapid and extreme fluctuations in blood-supply may give rise to many symptoms, varying according to the function of the part which is the seat of the disorder.

Such then, in brief, are the two physical conditions of the nervous system in neurasthenia, to which I have wished in the outset to direct your attention. Simple as they may be, I must insist that you clearly apprehend and bear them in mind.

Out of these grow other conditions of importance already alluded to, of which the more important are the following:

1. Loss of power, either physical or mental, or both. Such patients are unable to do or endure what they are able in health to do or endure.

2. Augmented sensibility either in the domain of the lower physiological sensibilities, or that of the emotions. In neurasthenia, patients are usually more easily affected than the same persons are in health.

3. Reflex excitability is greatly exaggerated, as a rule, in all

directions. This depends partly on morbidly increased mobility of the nervous system, and partly on a loss of that control which the higher centres exert over the lower ones, but especially which the highest part of the brain, which is the seat of the will, exerts over the lower centres in the brain, medulla and cord, the tendencies in which to reflex responses to the excitations which reach them from various quarters, the will centres control. This power of the brain for the control of the lower centres in neurasthenia, is exhausted in various degrees the same as other forms of nerve power, and hence the difficulty of control in such cases. Between the positive gain in reflex excitability in the central as well as more peripheral parts of the nervous system, and the positive loss of power for control, or of inhibitory function, reflex excitability is seen to be greatly augmented. Impressions reaching the spinal cord, medulla, or brain, either by the sensory nerves of the peripheral nervous system, or from the cortex of the brain above, from the seats of emotion, by the way of the fibres descending from the cortex to enter the cells of the basal system, or medulla, or spinal cord, are projected into reflex mechanisms more responsive than they are when healthy, and not only do they respond more readily to the impressions which reach them, but these impressions meeting less resistance, irradiate throughout the nervous system more freely than they should do normally, and create a disturbance more wide and more enduring, as the neurasthenia is more profound. This undue reflex excitability is shown in many ways: 1. In an abnormal tendency to involuntary muscular action. The type of this kind of manifestation of augmented reflex excitability is chorea, and almost on a par with it is hysteria. But it is shown in very many other diseases, as I shall try to show you before I am done with this course of lectures. 2. In abnormal increase of the cardiac and vaso-motor reflexes. This is a point of extreme importance in neurasthenia, and is one on which I must dwell to some considerable extent.

It is now known, as never before, that the vascular system sustains very close and important relations to the nervous system. The heart has not less than three kinds of nerves, functionally speaking, going to it, and not less than one kind going from it. Those going to the heart are the cardiac motor portion of the vagus (probably inhibitory), the accelerator nerves (Cyon, Ludwig,) from the cervical and dorsal sympathetic, and the vaso-motor nerves of the vessels proper of the heart. Aside from the more ordinary nerves of the heart, if not to be included with them, is the "depressor" nerve of the heart—a sensory cardiac nerve, which arises largely, it is probable, from the endocardium, and proceeds to the medulla, for important and widespreading reflex purposes.

Then there are the muscular arteries, especially before they terminate in the capillaries, which are, like the heart, supplied by nerves. They have, first, local apparatuses of ganglion cells, which probably receive fibres from, and emit fibres to, the vessels. Each vessel has, thus, its own independent nervous supply. But these local apparatuses are connected and co-ordinated by apparatuses, have a wider sweep of relations, and hence the larger, more central ganglia of the extra spinal portion of the so-called sympathetic nervous system. But, like the automatic and intrinsic apparatus of ganglion cells and fibres of the heart, which is connected by means of the vagus and accelerator nerves with the grey matter of the cord and medulla, so with these local extra-spinal nervous apparatuses of the vessels. They are freely connected with the grey matter of the spinal cord by means of fibres which pass out (from the cord) to terminate peripherally, not in the muscular tissue of the walls of the vessels themselves, but in the ganglia and ganglion cells of the peripheral vaso-motor mechanisms, which, in their turn, stand in immediate relation with the vessels. These vaso-motor nerves, which proceed out from the grey matter of the cord, probably, as just described, control the action of the peripheral apparatuses in which they terminate in much the same way, doubtless, as the vagus and accelerator nerves do the action of the intrinsic motor ganglia of the heart.

There may be, functionally speaking, even two kinds of vaso-motor nerves passing out from the grey matter of the spinal cord, as has been supposed—constrictor and dilator, comparable to the slowing and accelerator nerves of the heart.

But one thing is now certain: viz., that vaso-motor nerves are emitted from the cord—in all probability from its grey matter. It is true it yet awaits practical demonstration; but it has long seemed highly probable to my mind, that there is a tract of grey matter in the spinal cord out of which the spinal vaso-motor nerves arise, passing out chiefly by way of the anterior (motor) roots of the spinal and cerebral nerves. This supposed vaso-motor tract in the grey matter of the cord, if it exists (as I believe it does), must extend—differently from what many seem to suppose—the whole length of the cord, up into the medulla, side by side with the better known musculo-motor tract in the grey matter of the cerebro-spinal axis.

In the medulla, and perhaps in the basal system of ganglia, which I have described to you, this central vaso-motor tract culminates in a chief or supreme centre, toward which all vaso-motor centripetal impulses tend, and from which may issue in return those impulses which may by descending the spinal cord, and passing out from it at various heights, by the way of the fibres just described which pass out towards the vessels, affect thus the action of any or all parts of the peripheral vascular nervous apparatus. In this view the entire vaso-motor nervous system is summed up in the medulla and—perhaps—basal system. By this means the vaso-motor nervous apparatus is placed in close relation with the cardiac, the chief centres of which lie also in the medulla, so that these two parts of the grand vaso-motor nervous apparatus may affect each other or be affected by the same causes, so they may in general terms, have close relations in regulating the circulation of the blood.

Now the action of this great nervous apparatus (vaso-motor) is essentially the same as that of other parts of the nervous system. It acts in a reflex way. But from what sources come the excitations by which its action is excited or affected? In health in the case of the heart, it is probable that some impressions made in this organ, perhaps on its interior lining membrane by the blood, are conveyed to its motor ganglia, and these react through motor fibres on the muscular tissue in the walls of the heart. But beyond this, there must be fibres which arise from the heart, and which are for the purpose of conveying directly to the cord and medulla a knowledge, as it were, of the condition of the heart, for the purpose of securing reflex responses, either through the slowing or accelerator nerves, according as the heart needs to change its action, as it does from time to time. But can the action of the heart be changed alone in the manner described, by excitations starting in the heart itself, and returning to it through the purliens of its nervous mechanism as just described? May not the sources of excitation be extra-cardiac? Most certainly so in my opinion. The original seat of irritation may be the mucous membrane of the stomach, as it so often is, or of the bowel below, or it may be in the genito-urinary mucous surfaces, as happens in thousands of cases, or it may be in the skin, or it may be by way of the organs of the special senses, or more important yet, it may be in the cortex of the brain, which is the proper seat of mental action, whatever its kind; from any or all these sources, the excitant influences may start, which, when they reach the central cardiac nervous apparatus in the medulla, may excite it, just as certainly in a reflex way as it could be by impulses arriving at the same point from the heart itself. Hence it comes to pass that irritation of the stomach, or of the sensitive genito-urinary surfaces, or irritations of the skin, or excitation of the cortex of the brain, and of many other sensitive parts, give rise to disordered heart action, in various degrees and ways.

The same that can be said of cardiac nervous action, and reflex disorders of the same, may be said of the action of the vaso-motor apparatus proper. It may have, and has, its local limited disorders, independent of the action of the spinal cord and medulla. But it is more common by far to find, that the circulation of the blood in some part of the body, or even of the whole body, has been affected by means of influences exerted on the spinal cord, or its grey matter, and from this outward by the channel of the nerves which I have described to you as passing between the cord and the peripheral vaso-motor mechanisms, upon which the vessels immediately depend. According to the character of the influence which passes out from the cord, the vessels will contract or dilate, and hence admit more or less blood, in a given time, to the part, whatever it may be, the vessels of which are acted on.

The excitant influences affecting the action of the vaso-motor apparatus may come from any or all the sources already described, in relation to the heart: viz., from the vessels themselves, more or less directly, by way of any nerve of sense whether general or special, or from the brain above, the excitation in this case starting in its cortex, and descending its fibres to the motor and vaso-motor grey matter of the medulla and cord, in which many of its fibres directly terminate, and the action of which is thus excited—in any or all these ways may the central vaso-motor tract be affected, so as to emit the nervous influence, which may finally change in varying degrees the circulation of the blood in different vascular areas of the body, and in so doing, change the rate of nutrition of such parts, and by consequence, everything depending on the same.

Now this really important part of the nervous system shares with others in the loss of power and gain in reflex excitability, observed in neurasthenia, and hence the feebleness, excitability, and irregularity in action of the vascular system in neurasthenia. It is easily excited by impressions of a comparatively trivial character, either from the sensory *peripheral* nervous system, or any part of it, or from the brain. The circulation fluctuates under the slightest provocation, of course, under influences won from the vaso-motor nervous system. The heart responds by palpitations and irregular action with abnormal facility, and in this way the whole circulation is modified, at times, so as to produce the most alarming symptoms. In like manner the muscular vessels contract or expand with great facility and irregularity, so that rapid changes in the amount of blood in a part occur, thus giving rise to a legion of symptoms, according to the functions of the organ which may happen to be the seat of the fluctuation in blood-supply, and the action of which by such means may be changed. This fluctuation in blood-supply is especially likely to occur in the spinal cord and brain, giving rise in this way to many symptoms, as may be seen more in detail hereafter. Under this same head of fluctuation of the circulation, should also be mentioned changes of posture as a cause. In cases of neurasthenia, there is usually, if not always, a general diminution of vascular tonus. This makes it possible for changes in posture to produce sudden variations in blood tension, especially in and about the brain. Hence it comes to pass that such patients, if they suddenly rise to the sitting or standing from the recumbent posture, often immediately turn pale, experience vertigo, obscurity of vision, tinnitus, sudden change in the heart's action, confusion of mind, and at times syncope, and many other less striking examples of the same kind.

It is true, that symptoms produced in the way just described may depend, to a great extent in some cases and to some extent in all, on a *diminished volume* of the blood. But it more often depends on a loss of vascular tonus, and this depends on exhaustion (or neurasthenia) of the vaso-motor nervous system, which shares in varying measure the fate of other portions of the nervous system.

But I cannot now go farther into a study of the organic conditions which underlie neurasthenia. They are, I may repeat, a lesion of nutrition, and irregularity, and sudden fluctuations in blood-supply, especially to the cord and brain. Let these conditions be clearly had in mind. This being done, I will proceed to show you some of the more important *remote* causes or conditions of neurasthenia. This will be given in my next lecture.

ART. V.—THE PEDUNCULAR TRACTS OF THE ANTHROPOID APES.

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NUMBER I.

THE attempts of biologists to refer the origin of the human species to a progressive development from an extinct and thus far problematical anthropoid ape, have induced both the defenders and opponents of the doctrine of evolution to make a careful study of the existing human-like apes of Africa and Asia. It was but natural that those in favor of this doctrine should lay greater stress on the resemblances, and that their opponents should attach more weight to the differences found between man and his nearest allies; but, on the whole, this controversial turn of the discussion was exceptionally a source of profit: for it induced numerous observers to accurately study the debatable points, and thus enabled them to correct the errors into which the earlier anatomists had fallen.

It cannot be denied that the general result of these inquiries has been to verify the proposition of Huxley, that the differences between the anthropoid apes and the human species are less than the differences between the former and the quadrumana next below them in the scale. One line of defense after another has been erected by the opponents of the evolution doctrine, and one after another has been swept away! The organ of the mind was the last conservative stronghold, and here the contest waged most bitterly within a very recent day.

At first it was maintained that man alone had a considerable cerebral overlap; it was then demonstrated that not only the anthropoid but common apes had as considerable an overlap as some human specimens. Then it was asserted that man alone had a posterior cornu of the lateral ventricles, and a hippocamp at its floor. Now it is well established that not only the highest but the lowest monkeys—such as the lemurs—have both cornu and hippocamp, and that these characters are not even limited to the quadrumana. It was next claimed that in man alone is the island of Reil completely covered by overlapping convolutions; and when this was shown to be an error it was as gratuitously asserted, and for a time even accepted by the evolutionists, that man possessed, and the anthropoid did not possess, gyri on this area. The existence of such sulci and gyri in the brains of the chimpanzee and orang-outang, and even the non-anthropoid baboon is unquestionable.*

Unable to demonstrate any fundamental difference, observers like Bischoff have resorted to a comparative study of the gyri on the cortical convexity, and established diagnostic marks on the presence or absence of transition gyri, of an occipital operculum, or an inferior frontal convolution. As soon as the discussion took this turn, a number of monographs on the cortical gyri followed each other in rapid succession. Huxley, Barkow, Bischoff, Broca, Gratiolet, Hamy, Vrolik and Van der Kolk, Marshall, Rolleston, Turner, Vogt and Wernicke have dealt more or less exhaustively with the gyri of the chimpanzee and orang-outang, while Pansch, of Kiel, has now furnished that desideratum, a description accompanied by photographs of the cerebrum of the gorilla. Among the permanent results of these inquiries, our present perfected knowledge of the human gyri and their variations is the chief.

But perhaps the tendency of all these writers has been too exclusive, attaching an unduly preponderating importance to the gyri *per se.* If the relations of mammalian families were to be made even partially dependent on cortical resemblances, then the spider monkey, with his long prehensile tail and rudimentary thumb, would rank unpleasantly close to the human being; and Vogt † has shown with considerable humor that if the characters considered by Bischoff to separate man from the highest apes be the essential requisites of the human brain, then all that would be necessary were to melt the so-called "devil's ape" (Ateles Beelzebuth) and "Capuchin-ape" together to constitute the lord of creation!

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* "Contributions to Encephalic Anatomy, Part V.: The Organology of the Island of Reil," Journal of Nervous and Mental Disease, January, 1879. aut.

† Carl Vogt: *Lectures on Man.* English edition. Published by the Anthropological Society, London. 1864.

The anatomy of the cerebral surface has therefore been carefully studied; but the same cannot be said of the structures and relations of the peduncular tracts. Either no reference is made to the latter or a general surface description is given. Such structures as have no assignable functional importance, as, for example. the *corpora mammillares*, have formed the chief subjects of discussion, while important features, such as the relative degree of development of the pons, the pyramids, the restiform columns, the nerve nuclei and the relations of the trapezium have well nigh been overlooked. This neglect is the more remarkable, as Meynert * has shown in his suggestive monographs, which have appeared during the last ten years, that the relations of the peduncular tracts and basal ganglia to each other, present well marked differences in different animals, and that the superiority of the cerebral hemispheres in man is projected in a corresponding predominance of the pes pedunculi, the pons proper and the anterior pyramids.

I shall proceed to present the results of an analysis of the peduncular tracts of two anthropoid apes, and to compare their characteristics, step by step, with those of the corresponding human parts.

The series of specimens on which I have based this description were obtained from two chimpanzees, both males. One of these was perhaps the largest specimen which has been kept in captivity, and had, judging by the development of its genitals, arrived at the age of puberty. The brain of this chimpanzee, when divested of its membranes and fluids, weighed grammes 389.86; there was some atypical asymmetry of the hemispheres, which had probably been related to the mental idiosyncrasy observed during life; and to preserve this appearance the brain was kept intact. The other specimen was obtained from an infant chimpanzee, whose age may have been a little over two years. It died of tuberculous meningitis, and the preservation of its softened nerve centres implied the use of alcohol for the first few days, after which the peduncular tracts were immersed in bichromate of potash, and a series of over four hundred transverse sections was obtained—the sections ranging from the decussation of the pyramids to the altitude of the corpore mammillares. One hundred and forty of these sections were selected for preservation. The methods employed were the same as those described in my article entitled "Contributions to Encephalic Anatomy" (Part I.), published in the JOURNAL OF NERVOUS AND MENTAL DISEASE for October, 1877. For purposes of comparison, I had at my disposal thirteen more or less complete series of sections from human pedunculi, as well as the tracts of an orang-outang, for whose brain I am indebted to Mr. Hermann Dorner, Ph. D., who also furnished me the bodies of the two chimpanzees referred to above. Other series from lower monkeys, such as the cebus, semnopithecus macaque, and baboon, I owe to the courtesy of Mr. Conklin, Superintendent of the Zoological Department of Central Park.

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* "Ueber Unterschiede im Gehirnbau des Menschen und der Saugethiere." 1870. — "Beiträge zûr Kenntniss der centralen Projection der Sinnesoberflächen." 1869.—"Studien ueber die Bestandtheile des Vierhugels, soweit sie in den nâchst unterhalb gelegenen Querschnitten der Brucke gegeben sind." *Zeitschrift fur wissenschaftliche Zoologie*, XVII. Band. See also "Contributions to Encephalic Anatomy," JOURNAL OF NERVOUS AND MENTAL DISEASE, 1877-78-79.

Before giving the results of my comparative measurements, I shall describe the coarse anatomy of the parts in question, and then take up the features distinctive of certain topographical altitudes in their order.

It may be remarked here that there is a considerable difference between the tracts of the two chimpanzees. The elder one, whose hemispheres were remarked as being more richly convoluted than those of the younger animal, had, to judge from the plastic elevation, better developed pyramids and olivary bodies than the latter. The nuclear elevations were also better marked; this was probably due to the difference in age.

There is no doubt that if my measurements had been taken from the older chimpanzee they would have compared more favorably with the human measurements.

The ventricular contours were studied on the series of transverse sections taken from the younger animal.

At the junction of the spinal cord with the medulla oblongata, the central canal is a vertical slit, which is asymmetrical in some sections, showing a faint tendency to send out transverse diverticuli, and becomes linear and regular in the altitude of the lower pyramidal decussation. Before the middle of this decussation, the canal as such is obliterated by a loose mass of cells, which easily falls out of the sections and leaves a laurel leaf shaped gap; where the mass is intact it in some sections shows the openings of one or two irregular sinuous channels. At the commencement of the upper decussation the canal is once more open and remains so.

From this point on the central canal begins to be approached by the posterior fissure of the medulla oblongata, becoming at the same time more slit-like. It joins the posterior fissure just above the pyramidal decussation very gradually, and is at first connected with the latter by a mere slit enclosed by the walls of the posterior fissure. The central canal has entirely merged into the fourth ventricle in the region where the hypoglossal nerve roots become most distinct. The *alæ* of the posterior half of the fourth ventricle are but feebly marked, and just behind the striæ medullares acustici, there is no demarcation whatsoever. In this respect the medulla before us contrasts markedly with that of the human being, but it must be recollected that this chimpanzee died of tuberculous meningitis, and that the incipient hydrocephalus present, obliterated the contours at the floor of the ventricles. A pathological proliferation of the ependyma was observed, and to this doubtless was also due the obliteration of the central canal alluded to.

The anterior half of the fourth ventricle exhibits well marked and broad *eminentiæ teretes*, and a deep *fossa cœrulea*, which however shows no color as in the *adult* human being. In the region of the quintus origin, where the ventricle begins to undergo its gradual transition into the aqueduct of Sylvius, and is covered by the *lingula cerebelli*, it is seven-sided, the broadest side being under the lingula, two arched sides with a deep slit between them correspond to the *eminentiæ teretes*, the smaller sides being lateral. The lower of the lateral walls begins to diminish and finally disappears, so that in the region of the *velum* behind the origin of the trochlearis nerves the aqueduct becomes five-sided, the angle of the pentagon being directed downwards. Still further on the remaining lateral walls disappear and the aqueduct is now an inverted triangle, which gradually changes to an arrow-head shaped figure in the region of the posterior corpora bigemina. In the altitude of the anterior pair it becomes slit-like and vertical, continuing thus until it opens into the third ventricle, where it has become more three-sided again. The transverse section of the aqueduct exhibits a wavy contour, corresponding to a longitudinal striation, termed *pectunculus* in the human brain by Arnold.

At the posterior end of the fourth ventricle, a not very well marked obex is observed, which is exclusively composed of white substance. It is in man sometimes composed of white and sometimes of both grey and white substance.

Aside from the results of pathological alteration, the aqueduct, fourth ventricle and central canal correspond to the most common form of these parts in the human species. It is well known that in the latter, there are numerous variations, the general type as indicated is represented in the anthropoid ape also.

On examining the base of the brain, we are struck with its close resemblance to that of a child, especially is this resemblance marked in the peduncular tracts. Behind the thick and well marked chiasm, which is rather broader relatively than the human, we perceive a long and thin infundibulum, to which a vertically flattened hypophysis is attached, of the same color, shape, and relative dimensions as in the human subject. The corpora mammillares are prominent and distinct from each other, being separated by a deep notch.

The crura are almost cylindroid, they have a bold convex contour, and are not irregularly bulged out by deeper ganglia as in lower animals. The pons is massive, presenting a beautiful double sweep of its anterior outline as in man, with a median notch, beneath which a deep *fossa cæca* is found. The posterior outline is straight, and beneath it a similar cæcal depression exists, exactly as in the human subject. The fibres of the pons entering the cerebellum by its middle peduncle are mainly directed backwards. In the larger chimpanzee the antero-posterior diameter of the pons is 19 millimetres, the transverse, measuring between the outer contour of the large quintus roots, is 28 mm. The pyramids emerge from the pons as well-marked half cylinders, each pyramid singly measuring 5 mm. across, a slender *ponticulus* (Arnold) runs over them at the point of exit from the pons. At a distance of 2.5 mm. from the posterior pons-border, the olivary eminence begins, and from this point on the pyramids become narrower and flatter, to expand again without regaining their previous convexity at the lower extremity of the olivary elevation; further on they gradually become narrower and narrower until they terminate superficially as sharp points; the contour of the pyramids together with the median fissure between them resembles a steel pen much more than the ventricular "calamus scriptorius." The distance from the pons to this superficial termination of the pyramids is 20 mm., and the decussation itself is concealed in the depths of the anterior median fissure.

The olivary body is as prominent as in the human subject and bears the same relations to its surroundings, it is well demarcated, convex and *lobulated*, being divided into three or four imperfect lobes, exactly as in man. Its length is 11.20 mm., its width 5 mm. The width of the entire medulla at this point, measuring from the outer contour of one vagus root to the other, is 18 mm.

At the point where the medulla oblongata has undergone a complete transition into the spinal cord, the latter measures in the antero-posterior diameter 6.30 mm., in the transverse 10.2 mm. All these measurements were taken from the brain after it had been preserved for six months in alcohol, and are consequently smaller than the measurements taken from the recent specimen would have been.

The nerve roots present nothing calling for special notice, being in every respect identical with the human nerve roots. It is noteworthy that the diminution which the medulla undergoes from before backwards is gradual and even, not sudden as in the baboon, ateles and cebus.

Taking these parts as a whole and comparing them with lower animals on the one hand, and man on the other, we remark as points possessed in common by man and his anthropoid relative the massive character of the pons, especially in its anterior third. In the larger chimpanzee the height of the pons — by which I mean its measurement taken vertical to the surface — is 14 mm. In this respect man and the anthropoid contrast markedly with all other animals. A second feature is the bold contour of the pyramids at their point of exit from the pons, and the size and lobulation of the olivary eminence. The existence of a well-marked olivary eminence, I may state here, is not necessarily an indication of a well-developed olivary nucleus. Meynert has evidently had imperfect specimens at his disposal, when he states that the baboon has a rudimentary olivary eminence, and the dog none. The eminence is present in the latter, and is well pronounced in the former; and in ateles it is as distinct and of as plastic a contour as in man, though occupying a relatively smaller area. On the other hand, the seal, for which animal Meynert claims an olivary body as well-marked as in the highest apes, has an eminence, it is true, but the olivary nucleus itself is not better—nay, not as well—developed as in the bear. The elephant has an excellently marked olivary eminence: but the olivary nucleus is of a very simple structure. Consequently the height to which the olivary *eminence* attains is no criterion for the development of the *nucleus* itself. A bulky but unconvoluted nucleus may be associated with as high an eminence as a very intricately convoluted one, and an eminence may be present where the nucleus itself is so small as not to have had any influence in producing it, so that the eminence may be entirely composed of white substance.

Even in monkeys, with as high a hemispheric development as semnopithecus, macacus and ateles, the anterior half of the medulla oblongata narrows down very suddenly in joining the posterior half. The suddenness of this reduction in mass is, as Meynert has shown, most marked in lower brains, and is therefore still greater in the dog and rabbit than in the monkeys mentioned. It is due to the presence in the anterior half, and absence in the posterior half, of a considerable amount of molecular nerve substance, as the same author has shown.

In the chimpanzee this animal feature is absent.

There is no trapezium in the chimpanzee: the predominance of the pons conceals the fibres of that body as completely as in the human being. Immediately behind the pons, on either side of the anterior pyramids, is a deep depression, as in the human species, and in this area no trapezium fibres can be detected.

Whether man and the anthropoids are the only animals in which the trapezium is concealed, I leave an open question. The same condition *seems* to be present in ateles.

The abducens and facial nerves make their exit from, around and through the posterior pons border, as in man. The relative position of these nerve roots to the pons shows that the latter extends as far posteriorly as the human pons. This is further shown by the distance between this border and the anterior extremity of the olivary eminence, which is relatively the same in both instances.

It must be here emphasized that the anterior end of the olivary eminence does not correspond to the anterior end of the olivary nucleus. The former does not reach the pons in any brain: the latter extends a considerable distance behind the pons fibres in the human being; its relations in the chimpanzee we will yet discuss.

Comparing the base of the chimpanzee's peduncular tracts with those of a well developed human brain, we observe that the pons is slightly less voluminous, and the pyramids flatter between the olivary bodies. There are, however, as great differences between the pons area of different normal human brains as between the lowest of the latter and the chimpanzee. Particularly in its anterior half does the latter's pons resemble the same part of certain human brains—in depth especially. On the other hand, I have not yet seen a human brain in which the pons was so thin on its *posterior* border as in the ape before us.

As to the anterior pyramids, these I have frequently seen to be as flat in man as in the anthropoid. As a rule, however, even here the notch separating the olivary eminence from the pyramid was deeper; but in one negro and in an epileptic imbecile I found, also, this as faintly marked as in the ape in question.

Taking the *ensemble* of the chimpanzee's tracts, whether as to their relative dimensions, the angles of incurvation dependent on the basi-cranial axis, or their general contour, we must say that they resemble the corresponding human parts much more closely than those of the baboon resemble those of the chimpanzee. The latter possesses several other features present in none of the non-anthropoids: that is, the massive prominence of the anterior extremity of the pons, its near approach, when the brain is regarded in situ, to the infundibulum (topographically), and the direction of the crura, which is at so steep an angle that, unless the parts be artificially stretched, they are almost concealed from view. These, like the corresponding features in man, are related to the basi-cranial conformation, and are therefore still more pronounced in the human species.

In the orang-outang the parts are very similar to those of the chimpanzee. We notice, however, a far more human-like appearance of the pons. While, on the one hand, its mass is not very much larger, it is more evenly distributed, and the posterior edge is not as attenuated as in both the chimpanzees examined. The demarcation externally, between the olivary elevation and anterior pyramid, is as imperfectly marked as in the other anthropoids; but on the lateral aspect of the medulla, it juts out much more prominently than in the latter. The disposition of the nerve roots is the same as in man, and the trigeminus does not appear to be as large as in the chimpanzee. The crura cerebri are more convex, and are separated by a deeper inter-peduncular notch than in the other human-like ape.

The dimensions measured on the hardened and shrunken specimen are as follows:

Width of chiasm	13	Millimetres.
Width of optic tract	5	"
Thickness of optic tract	3	"
Length of exposed part of crus cerebri	6	"
Width of exposed part of crus cerebri	12	"

Length of pons varolii	18.5	Millimetres.
Width of pons at quintus roots	28.	"
Length of anterior pyramids	17.5	"
Length of olivary eminence	13.	"
Distance of same from edge of pons	1.8	"
Diameter of medulla, measured from inner aspect of one auditory root to other	16.	"
Same at decussation	10.5	"
Diameter of pyramids at emergence from pons	6.3	"
Same in middle olivary altitude	5.5	"
Same, three millimetres below olivary eminence	4.5	"
Length of fourth ventricle	20.	"
Greatest width	15.	"

The elevations at the floor of the fourth ventricle are much better marked than was described to be the case with the younger chimpanzee. The *alæ albæ medialis* and *alæ cinerea*, as well as the obex, are very prominent.

We will now proceed to study the series of transverse sections from the younger chimpanzee.

In the altitude of the first cervical nerve roots, the transverse section shows the same general distribution of white and grey matter as in man. The anterior cornu is broad and contains from thirty to forty multipolar cells, the processi reticulares are thin but numerous, and the neck of the posterior horn is almost entirely interrupted by vertical strands. The gelatinous head of the posterior horn nearly touches the periphery of the cord, and there are but few ascending quintus bundles visible. The decussation of the pyramids is still in progress, and coarse decussated bundles interrupt the continuity of the grey substance here and there, while fibres are seen emerging from the inner aspect of the anterior columns as the transition from the last pyramidal to the uppermost anterior commissural fibres. The area of the posterior columns is very large, this is a markedly human* feature. If the entire area of the section be taken as 1.0000, the human will be found to be .2235 and upwards, in this chimpanzee it is .2219, in the cebus monkey .1382.

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* The area of this column in the upper cervical region is very small in reptiles and amphibians.

There is a greater discrepancy in regard to the columns of Türck; none of my human specimens show less than .0369, the chimpanzee measures .0295 and .0300, the cebus monkey .0243.

In the lateral columns, the human being measures .2710, the chimpanzee .1955, and upwards in other sections, the cebus .1463. Taking into account that the latter measurement is in part so high because of the deficiency of other tracts, and the presence of much molecular nerve matter, the real proportion of the three cases would be: human .2710, chimpanzee .1955, cebus .1140. It is well known that these tracts vary extremely in the human being. The section as a whole could be mistaken for a human one readily, and in distinction from those of lower monkeys, exhibits a human-like proportion of the diameters. Taking the antero-posterior diameter as 10, the transverse in the human being is 11–13, in the chimpanzee 11.3, in the cebus .15.

The area measurements obtained were made in the following manner: A drawing of the section was made whose correctness was ensured by repeated measurements. A glass plate with numerous small squares ruled thereon was then placed over the drawing and the number of squares and their fractions gave the required relative areas. Although the last two decimals are so fractional as to be of no special importance, yet as they were obtained in the computation I have given them:

TABLE A.

	Area of Lateral Column.	Area of Posterior Column.	Area of Turck's Column.
Man	.2710	.2235	.0369
Chimpanzee	.1955	.2219	.0295
Cebus	.1463	.1382	.0243

As we pass through the series we notice the same metamorphosis of grey and white matter as in the human medulla. The ascending quintus root is remarkable for its size, but does not exceed the limits of the human range, the caput gelatinosum is folded as in man, while in lower animals it is a simple oval mass (even in cebus). The histological characters are essentially human, and if I were to describe them and the coarser anatomical characters in detail, it would but lead to a repetition of the well known facts of human anatomy. It is noteworthy, however, that the anterior pyramids are proportionately defective as compared with those of man.

The fibres of the motor decussation occupy a much larger area in man, and as the pyramidal fibres which are still vertical in this altitude are also more voluminous, it results that, with the same configuration of the grey substance, the vertical (antero-posterior) diameter of the human medulla is relatively greater. Taking the transverse diameter as a standard of 10, the antero-posterior is 9.2 in man, 8.8 in the anthropoid.

The nucleus cuneatus is larger in the chimpanzee, while the nucleus gracilis is of the same dimensions and as scattered as in the latter.

The measurements of sections from the altitude of the superior decussation, taking the entire area as 1.0000 are as follows :

TABLE B.

	Area of Pyramid.	Area of Posterior Column.	Area of Gelatinous Cornu.	Area of Nucleus Cuneatus.
Man	.3098	.1032	.0647	.0283
Chimpanzee	.2384	.1846	.1000	.0384
Cebus	.1093	.1406	.1718	.0468

While at the level of the first cervical nerve roots the posterior columns of the anthropoid ape were equal to those of the human being, we now observe a marked difference, the chimpanzee possessing a much larger area in the present altitude. Now, this difference must be due to some quantitative difference in the distribution of the fibres, and I find strong reasons for considering it as complementary to the deficiency of the chimpanzee's pyramids.

Since the anterior pyramids are known to obtain a large portion of their fibres from the lateral columns of the cord, one might be inclined to refer this difference of the two pyramids to the difference between the respective lateral columns. But I find this explanation inadequate. Undoubtedly a considerable part of the discrepancy can be so accounted for, but not all. I find the fibres of the superior fine-bundled or sensory decussation to be remarkably fewer in the ape than in man. Consequently, if we suppose that less of the posterior column area decussates than in the human being, we shall understand why the posterior column remains larger than, while the pyramids fail to become as large as, the human.

That a relatively greater portion of the fibres of the posterior column in the chimpanzee must terminate in the nucleus cuneatus and gracilis will become evident as we proceed.

Apparently, the cebus monkey occupies a position intermediate between man and the higher ape; but if the originally defective character of the posterior columns (table A) be taken into account, it will become evident that the fibres of the pyramidal category are still more poorly represented in the posterior columns, while a relatively greater proportion terminates in the funicular nuclei.

The higher sections exhibit the origin of the olivary nucleus. In the similarity, nay, almost identity, of the shape, dimensions and relations of this body I find one of the strongest resemblances to the human being. As my views on the origin of the olivary body, and especially the relations of the internal accessory olive, differ from those accepted by the majority of writers, I refer the reader to an earlier article * in the JOURNAL OF NERVOUS AND MENTAL DISEASE for an explanation of the doubtful points.

The internal accessory olive is first struck on section in the upper half of the sensory decussation; its appearance is exactly like that of the human being; and, like the latter, it seems to rise out of the reticular formation at the point where the latter abuts on the pyramidal area. In the cebus, this body does not appear until the sensory decussation begins to undergo the transition to the restiform crossing, and differs markedly in other respects to be alluded to.

As in man, the accessory olive joins the vertical cell mass, developed on the outer border of the remnants of the anterior funicle; and at the same altitude in which, in man, the nucleus dentatus (body of olive) occurs, it also is seen in the chim-

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* "The olivary body in man, the anthropoids and higher animals." October, 1878.

panzee. First, a single dentation of this nucleus is struck in the altitude of the beginning restiform crossing, and in the next sections the olive proper shows five dentations. In man these dentations are usually so much acuter than in the chimpanzee that they are capped off separately by the knife. In my specimens from the chimpanzee, this occurs with one dentation, but on one side only.

Where five dentations are struck, two of them show secondary foldings.

I will now follow up the appearance of the olive on successive sections, and dispose of this part of the subject before resuming the description of the entire sections. In the altitude where the central canal communicates with the posterior fissure by a slit, the olivary nucleus proper shows an upper richly and sharply crenulated line, with three fundamental folds. The two inner are as yet simple, but the outer is dendritic, and sends out a long process, which is turned on itself and reflected along the lower outer border of the main part ot the olive. This latter is simple, showing only two folds. Where the lower hypoglossal roots appear, the nucleus dentatus has five main dentations; but, reckoning all the foldings, there are from ten to thirteen on the entire contour. In these sections the external accessory olive is faintly indicated, while the internal sends from its upper end a sharply recurved process, which, as we pass up in the series of sections approaches the lower lamina of the nucleus dentatus.

In the region where the posterior end of the fourth ventricle is struck, the latter body is of so complicated a structure that what I term primary dentations are no longer distinct, being split up by secondary folds: there are seventeen folds in all. This number falls to fifteen and thirteen in the next section, but in the altitude of the vagus roots rises to twenty-one on one side, nineteen on the other. At the lower end a number of folds are so sharp as to have separated from the main body of the nucleus in one section. The recurved fold of the inner accessory olive has nearly joined the main nucleus, and the outer accessory nucleus is now well marked, but is separated from the upper lip of the *hilus olivæ*, as in man.

Twenty-one* folds is the highest number reached; it begins to sink slowly from the time that the hypoglossal nucleus diminishes, while the internal accessory olive entirely disappears by the time the auditory root level is reached. Here there are fifteen folds. Now this number rapidly sinks; within the full breadth of these roots it becomes seven, then four, and remains so to above the root. At the same time the outer accessory olive diminishes and is fused with the upper lip of the hilus. The area of the nucleus dentatus has become immensely reduced, and while at the posterior pons border there are still three folds, enclosing a completely circumscribed cavity, at the point where the abducens roots (enclosed by pons fibres) begin to enter the medulla proper, a single mass surrounded by a molecular halo, and without a central space, is all that represents the olivary body. Before the abducens facialis field is reached it has entirely disappeared.

The following table will give a better idea of the differences between the human and anthropoid olive than any description:

TABLE C.

Altitude.	MAN.†	CHIMPANZEE.	CEBUS.
1.	Internal accessory nucleus appears.	Internal accessory nucleus appears.	No trace of olivary nuclei.
2.	Nucleus dentatus appears faintly.	Nucleus dentatus appears faintly.	Internal accessory olive appears.
3.	Nucleus dentatus, in five distinct masses.	Nucleus dentatus in two distinct masses.	Nucleus dentatus faintly indicated.
4.	These are reduced to three masses, each with three or four secondary folds. External accessory olive absent. Internal accessory olive well developed.	7 dentations on three fundamental folds. External accessory olive absent. Internal accessory olive well developed.	Nucleus dentatus a simple triangular area. The external accessory olive appears, and is united to the internal accessory olive for a short distance.
5.	Nucleus dentatus in *a* has 13 folds; in *c*, 21. External accessory nucleus faintly indicated.	Nucleus dentatus has 13 folds. External accessory nucleus faintly indicated.	Nucleus dentatus has two folds. External accessory nucleus in full development.

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*In a previous publication referred to I have given fifteen as the number. This was before I had examined an entire series of specimens.

†In this and the following table, the letters *a*, *b*, *c* and *d* refer to different individuals, sections from whose medullas were selected to show the range of difference in man.

Altitude.	MAN.	CHIMPANZEE.	CEBUS.
6.	Nucleus dentatus has 17 folds in *a*.	Nucleus dentatus has 17 folds.	Nucleus dentatus, 2 folds.
7.	Nucleus dentatus has, *a*, 19; *b*, 24, and *c*, 28 folds. External.	Nucleus dentatus has from 13 to 15 folds.	Nucleus dentatus, 2 folds. The internal accessory olive begins to disappear.
8.	Nucleus dentatus has, *a*, 18; *b*, 19 folds. Internal accessory olive begins to disappear.	Nucleus dentatus has 21 folds. Internal accessory nucleus well developed.	Nucleus dentatus, 2 folds. Internal accessory nucleus absent.
9.	Nucleus dentatus: *a*, 16; *b*, 18; *c*, 24 folds. Internal accessory nucleus almost absent.	Nucleus dentatus 17 folds. Internal accessory nucleus begins disappearing.	Nucleus dentatus, 2 folds. Internal accessory nucleus remains absent.
10.	Nucleus dentatus: *a*, 12; *c*, 23 folds. Internal accessory nucleus absent.	Nucleus dentatus has 15 folds. Internal accessory nucleus absent.	Nucleus dentatus has 3 folds. External accessory nucleus begins disappearing.
11.	Nucleus dentatus has, *b*, 9 folds. No accessory nuclei present.	Nucleus dentatus has 7 folds. No accessory nuclei present.	Nucleus dentatus a single mass. No accessory nuclei present.
12.	Nucleus dentatus, in *b*, 9; in *c*, 13 folds.	Nucleus dentatus has 4 folds.	All the olivary nuclei absent.
13.	Nucleus dentatus has, in *c*, 7 folds.	Nucleus dentatus a single round mass.	As above.
14.	All the olivary nuclei absent.	All the olivary nuclei absent.	As above.

In order that the reader may be able to compare herewith the dimensions of the area of the nucleus dentatus, I have compiled the following table, in which the whole area of the medulla is taken at 1.0000, the standard figure which applies in this essay to the total area of sections in any altitude :

TABLE D.

	Altitude 5.	Altitude 7.	Altitude 10.	Altitude 12.	Altitude 13.
Human, (*a*)	.0962	.1361	------	.0454	.0206
" (*b*)	.1110	------	.2106	------	------
" (*c*)	.1406	.1458	.1428	.0610	trace
" (*d*)	------	------	.0554(?)	------	------
Chimpanzee	.1006	.1270	.1384	.0240	.0160
Baboon		.0778	------	------	------
Cebus	.0240	.0705	.0526	absent	absent

In these tables altitude 1 refers to the latitude of the sensory decussation; 2, to the beginning restiform decussation; 3, the obex; 4, the same higher; 5, the lowest hypoglossal roots; 6, the hypoglossal nucleus in its full breadth; 7 and 8, higher altitudes of same; 9, the disappearing hypoglossal nucleus; 10, the *striæ medullares;* 11, the full auditory root; 12, the lower border of the pons; 13, two millimetres above this border; 14, the abducens roots where seen in their full length. It is needless to add that in the cebus altitude 12, 13, and 14, correspond to the trapezium and not to the pons.

While the olive is so closely similar to the human in its dimensions and shape, it is exactly like it in its histological structure; the thickness of the olivary lamina is, however, somewhat less, bearing a proportion of from three-quarters to seven-eighths of the thickness of the human olive. In lower animals, even in the Old and New World monkeys, the internal accessory olive bears an inverse ratio to the nucleus dentatus; where the latter is rudimentary, the former is massive, and absolutely in all these animals, while the nucleus dentatus is far inferior to the human, the internal accessory is superior. In the chimpanzee the latter is as attenuated as in the human being.

The restiform decussation is not so extensive in the chimpanzee as in man. The fibres are fewer, though their arrangement is the same. This fact bears an evident relation to the deficiency of the restiform column which is observed in higher altitudes, and regarding which there is a greater gap between man and the chimpanzee than there exists in regard to the anterior pyramids.

As we progress upward through a series of sections of the medulla, we observe that the funicular prominences are not as bold as in man, while the ventricular ponticulus is somewhat thicker, but when we proceed to measure the various diameters we will find their mutual proportions strikingly human-like. It may be accepted as a law in cerebral anatomy, that the more voluminous the cerebrum and cerebellum (for both, contrary to a popular opinion, keep pace to a certain extent) the greater will be the vertical diameter of the medulla; and of the two transverse diameters, one being taken through the restiform, and the other through the greatest width of the olivary region, the former will preponderate as we pass up in the scale.

In the following table, these dimensions have been arranged in various successive altitudes, the numbers of the latter corresponding to those in table C. The dimensions are given in millimetres :

TABLE E.

Altitude.	1.	4.	5.	6.	8.	10.	12.
Height of Raphe in Man	6.	$7.^{5}$	8.	$9.^{6}$	13.	14.	$12.^{3}$
In Chimpanzee	$4.^{5}$	$4.^{7}$	7.	8.	10.	$9.^{5}$	$9.^{8}$
Height of Medulla in Man	14.	$17.^{5}$	17.	$15.^{5}$	$17.^{5}$	18.	17–19
In Chimpanzee	$11.^{2}$	11.	12.	12.	$12.^{2}$	$12.^{2}$	13.
Transverse diameter through Restiform region in Man	15.	17.	18.	18.	23.	25.	25–29
In Chimpanzee	13.	$13.^{5}$	$14.^{5}$	$15.^{5}$	16.	$19.^{2}$	$19.^{5}$
Transverse diameter through Olivary region in Man	11.	$14.^{5}$	$16.^{5}$	16.	18.	19.	$14.^{5}$
In Chimpanzee	10.	10.	12.	13.	$12.^{5}$	12.	9.

In this table we observe that the raphe progresses in about the same ratio in man as in the chimpanzee, and that in both the greatest increase is in the same altitudes of the upper hypoglossal roots and the striæ medullares (8–10).

The total height of the medulla does not increase as rapidly as in the human being, though it reaches its maximum at the same point (12). From this it follows that the further we pass from the decussation to the pons, the flatter does the chimpanzee's medulla become as contrasted with that of man ; this approximation to the lower animals is however still more pronounced in the baboon and lower monkeys.

While the transverse diameter taken through the restiform region maintains the human ratio and proportion, that taken through the olivary body does not progress as quickly, and reaches its highest point (6) earlier than in *most* human medullas (10) ; this latter feature corresponds to the observation deducible from table C, namely that the point where the human olive *usually* shows the most intricate folding is on a higher altitude than the corresponding point in the chimpanzee, or rather that the intricacy of the dentations of the olive as well as its dimensions fall more rapidly in the anthropoid than in man (table D).

If we now suppose the total vertical diameter of the medulla oblongata to measure 100, and compare the proportions which the two transverse diameters bear to this standard in man, the chimpanzee and two lower monkeys, selecting the same altitude (8) of the hypoglossal roots in each, we obtain a result strikingly confirmatory of the correctness of the final conclusion to be announced :

TABLE F.

	Man.	Chimpanzee.	Baboon.	Cebus.
Vertical Diameter	100.	100.	100.	100.
Diameter through Restiform Columns	132.	131.	142.	133.
Diameter through Olivary Eminence	102.	102.	100.	96.

In higher sections the discrepancy between different human individuals in regard to these proportions becomes very great: in one medulla the flatness appears greater than even in the chimpanzee, but in reality this was an absolutely high medulla, with such immense restiform columns that the vertical diameter was relatively reduced. Within the area of the auditory roots, the proportion of the chimpanzee's bi-restiform diameter to the vertical is 150 : 100, in man 147 to 170—100, in the cebus 180 : 100 ; this latter measurement is dependent on the actually great reduction of the vertical diameter, due in its turn to the defective pyramids, and the real discrepancy is much greater than the figures show, on account of the extreme reduction of the restiform column.

On comparing a section from the chimpanzee's medulla, taken through the lowermost hypoglossal roots, with a corresponding section from the human, we notice, first, that the contour of the olivary eminence is less marked ; that the angle at which the internal accessory olive is bent (which is nearly a right-angle in man) is obtuse ; * and that the fibres of the pyramids, which in man are already cut obliquely, on account

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* This angle is the wider the lower we go in the scale; as the lower side of the angle necessarily follows the contour of the pyramid, it must become more and more driven up as the latter rises in development.

of the approaching decussation, are still to a slight extent vertical. The hypoglossal nucleus shows the same number and arrangement of the nerve cells found in man, and this is illustrative of a statement made on a previous occasion, that the innervations of speech in the human being do not require a more complex development of the lower automatic centres than is found in speechless animals. From this it follows that the same amount of grey matter can be under a lesser or greater multilocular subjection to co-ordinations and associations pre-existing in the highest centres: the nerve nuclei seem to follow in their mass—not the complexity of the function they subserve, but the size of the area to be innervated from them.

The proportions of the most important areas to the entire area are as follows:

Area of pyramid	.1111
" internal division of reticular field	.0432
" ascending V. root	.0092
" nucleus hypoglossi	.0308
" restiform remnant	.0493
Proportion of pyramid to internal division of reticular field (taken as 1.0000)	2.5706

In the higher series of sections we observe the same distribution of nerve nuclei, and as large a fasciculus teres as in man. The ascending quintus root is more crescentic, the human more oval in this region. Another difference is that the *stratum zonale* is half as thick again as that of the ape. This is, doubtless, to be interpreted as connected with the relative deficiency of the chimpanzee's restiform column, as it is well known that the *stratum zonale* derives its fibres from this column. The measurements are as follows:

TABLE G.

	Man.	Chimpanzee.
Proportion of anterior pyramid	.1555	.1111
Proportion of interior division of peduncle	.0666	.0761
Proportion of pyramid to internal division of peduncle; the latter taken as 1.0000	2.3348	1.4599

I find, on comparing a number of human medullas, that in the altitude of the middle olivary region the anterior pyramids vary from .1050 to .1750; in the same sections the chimpanzee has .0825, the cebus .0500.

Where the human restiform column measures .2190, that of the chimpanzee is but .1310. This discrepancy becomes more and more manifest as we proceed towards the cerebellum. Probably the lesser amount of zonal fibres and the rudimentary condition of the *nuclei arciformes* is hereunto related.

The auditory nerve root has the same intra- and extra-medullary nuclei as in man, and as in the latter, the fibres of the *striæ medullares*, or the superficial root fibres, extend to the raphe. Higher up there are seen fibres of this nerve running through the reticular field to the raphe, and for which, in the case of man, I suppose they represent a raphe projection to the cerebrum, differing with Meynert, who considers them to be a decussated set of cerebellar radicles.

In these altitudes the pons fibres begin to clasp the pyramids, a little asymmetrically, and include the emergent abducens nerve root. Now, while the pons, as stated in the macroscopic description, extends entirely as far downwards as in man, and therefore one essentially human feature is characteristic also of the anthropoid, yet there is a great discrepancy in the amount of pons fibres struck at the lower edge of this body. These are from three to four times as voluminous in man. As we proceed upwards, however, this discrepancy becomes gradually reduced. At one point, in fact, the chimpanzee reaches a higher figure than one human specimen.

TABLE H.

Proportions of pons to entire area of transverse section, exclusive of those portions which are properly cerebellar in their topography.

	Man.	Chimpanzee.	Cebus.	Dog.
In region of emerging abducens root	.2051	.0588.	none.	none.
In region of lower facial nucleus	.3710	.2870.	trace.	none.
In region of common abducens facial nucleus	(*a*) .4871 (*b*) .6470.	.5009	.2273	none.
In region of lingula cerebelli	(*a*) .7276 (*b*) .8000	.6923	.2980	.1190

In the region of the trochlearis roots the discrepancy between the human and anthropoid pons again manifests itself, but to a much less degree than in the lowest altitudes.

The brachium conjunctivum (*processus e cerebelli ad cerebrum*) bears the same ratio to the entire tegmentum that it does in man, and with this we find further on a well developed nucleus tegmenti, which rivals the human in size and is of the same histological structure.

We now have arrived at that region in which, according to Meynert, the proportions of the fibre masses subservient to the intelligence can be best ascertained, as contrasted with the fibres and ganglia concerned in automatism. It is well known that the *pes* or *basis* of the crura stands out more or less prominently in different individuals: in some it is flat and broad, in others narrow and high, and in still others as broad as in the former and as high as in the latter. This third condition I have found especially associated with well convoluted hemispheres, although I do not mean to imply the converse, that well convoluted hemispheres always must be associated with a large *pes pedunculi.*[*]

Here the chimpanzee occupies a rather intermediate position, and here is the first point in which a marked histological difference from man is noticeable.

The cells of the substantia nigra are imbedded in a molecular mass, which encroaches on the area of the pes pedunculi by sending out anastomosing processes, which isolate the peduncular fibres that adjoin this substance. In the chimpanzee this substance is at least twice as voluminous as in man, and as in addition to this encroachment on the pes pedunculi, its whole area is smaller, its absolute bulk is below that of the human being, though on the other hand far above that of lower animals. I estimate it as follows:

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[*] I believe that many insanities, as well as many conditions allied to insanity, such as the hysterical and "blue-stocking" propensities, may yet be traced to an inadequate projection of a well developed hemisphere. I have announced a similar view in an unpublished essay on the Somatic Etiology of Insanity. The development of powerful peduncular tracts demands certain proportions of the base of the cranium, as a *conditio sine qua non*, and as these latter proportions are more apt to be deficient in the female sex, this may explain the preponderating prevalence of the two conditions named in that sex. It is certainly suggestive in relation to this question, that males suffering from analogous forms of mental perversion, should have similar peculiarities of the cranium. In the female these peculiarities are normal, and exercise a prejudicial influence only when the hemispheric development exceeds the healthy average ; in the male they signify a tendency to degeneration, a lack of harmony between cerebral and cranial development, resulting from the enervating influences of city and sedentary life. A good physique, associated with a cranium whose ridges are prominent and whose foramina and inner recesses are well marked, is rarely if ever co-existent with the mental conditions referred to.

In man, from .3128 to .5219.

In chimpanzee, .2860.

In cebus, .1100.

In dog, .0632.

The nucleus tegmenti is, if anything, larger than in man, is as sharply demarcated and (as previously stated) of the same structure as in the human being.

The interpeduncular ganglion, which is large in the rabbit, dog, and other lower animals, is rudimentary as in our own species. The ganglion Sœmmeringii is rich in large multipolar cells, in a few of which there is a faint and doubtful indication of pigment. The central tubular grey matter and other masses are identical with the human, the corpora quadrigemina are however larger; I should say relatively to the human they stand as nine to eight.

In altitudes higher than the corpora mammillares I have not been able to carry on the investigation on account of the fragility of the tissues.

Summing up our results, we find first that in respect to the peduncular tracts, the anthropoid apes stand nearer to the human species than any other animal yet examined.* Secondly, that the differences between man and the anthropoids are less than those existing between the latter and the baboon, macacus, cebus, lagothrix, semnopithecus or cynopithecus.

I leave it an open question whether the ateles may not approximate the chimpanzee in regard to the olivary body, while I can state that it is far below the latter in the development of the pons.* With reference to the gibbons, which animals I do not class among the anthropoid apes, but intermediate between the latter and semnopithecus, I have no specimens for comparison. Judging by a figure from Bischoff, in which the peduncle is represented as cut through, I should suspect them to be nearer to the ordinary Old World apes than to the anthropoids in respect to the basilar tract.

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*In addition to using the measurements and diagrams of animal pedunculi, furnished by various authors, I have measured the tracts of numerous monkeys, the elephant, seal, bear, and other animals whose hemispheric development is not as great as in the three named.

In the development of the nucleus dentatus of the olive, in the preponderance of the pes pedunculi, the vertical pons fibres and the anterior pyramids, man and the anthropoids *together* possess characters separating them from all other animals. In the reduction of the molecular grey matter, by which the area of the strands connected with higher automatic and intellectual centres is inversely increased, we notice another striking parallelism between the two.

As one result of our inquiry, which may aid in solving some of the difficulties of human cerebral anatomy, I would point to the fact, that the restiform column of the chimpanzee is far inferior to that of man, while the brachium conjunctivum with its nucleus is as large or larger. In the first place this is confirmatory of the older supposition that the inferior and superior cerebellar peduncles are independent systems; in the second, it shows that the projection of general sensation through the upper peduncle (brachium conjunctivum) as well as of motility direct, is excluded, therefore these parts can be subservient only to associating, or to vaso-motor influences.

If we were to represent the average development of the higher (hemispheric and cerebellar) tracts of the human being as 100, the chimpanzee would rank about 75, the baboon at 40, the cebus at 25, the dog at 7½.

One point observed, the close approximation of the area ot the posterior columns in the chimpanzee and man, as contrasted with the great inferiority of these columns in lower animals, opens a question as to the relation between the development ot the posterior columns and the hemispheres. Our results corroborate Meynert's statement, that the superior pyramidal decussation is derived from these columns (against Flechsig), and the existence of a path of conscious sensation from the posterior columns to the posterior part of the internal capsule, is still to be maintained.

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* Although the trapezium seems to be perfectly covered.

Now, the development of the higher intellectual faculties rests in great part in the readiness and complexity of sensory appreciation, therefore *cæteris paribus*, the area of the posterior spinal funiculi in the cervical region would, like the area of the pes pedunculi, be an index of cerebral development.

The remarkably rudimentary condition of these columns which I have observed in turtles, frogs, the menobranchus and snake,* as well as the gradual rise in their area as we pass from these through the lower to the higher mammalia, is strongly in favor of the above view.

In many respects the present article is merely a preliminary communication, and I shall therefore refer only briefly to one other result which was obtained. If, of a series of sections from a human medulla, two be selected—one just above, the other just below the roots of the hypoglossal nerve—a considerable difference will be found in regard to the area of the anterior pyramids. This difference is evidently due to fibres which have been given off through the raphe to the hypoglossal nucleus, placing the latter under the cerebral control. In the chimpanzee this difference is not even approximately as great, a fact which furnishes in my mind a more logical explanation (anatomically) for the absence of speech in the anthropoid than the popular but fallacious reference to the presence or absence of an inferior frontal convolution, or of gyri on the island of Reil.

In this single respect the human peduncle stands alone, and a chasm separates it from all others ; but he who has studied the course of development followed by the cerebral fibres, will recognize in the rectilinear strands of the *raphe medullare* that potentiality of the speech function which our pithecoid ancestry must have possessed, and in no higher grade than the existing anthropoids, before this function became actually established.

While this latter inference is made in accordance with the prevailing views as to the ancestry of man, I refrain from pursuing a theme which, however interesting, enters on the domain of the speculative.

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* Also noticed in the specimens from the alligator demonstrated by Dr. Mason before the American Neurological Association.

The advocates of the doctrine of evolution will perhaps be able to formulate some conclusions on the facts of this paper which will support their views in regard to the relations of man to his problematical ancestry, as well as to the collateral descendants of the latter.

Reviews and Bibliographical Notices.

I.—ASYLUM REFORM IN THE STATE OF NEW YORK.

Report on the State Charitable Institutions of New York. Communicated by the Comptroller to the State Senate, April 9, 1879. Albany: *The Argus* Company. 1879. 60 pages.

In his annual report for the year 1878, to the Legislature, the Comptroller presents the clearest proof that "reform upon the canals has been real," by a comparison of their cost to the State under the old system of management by three commissioners, and the new one of a single superintendent of public works. It is enough to say, in this place, that although the new system had been in operation only one-half that year, the cost of the canals for the year was $310,580.71 less than in 1877. The economical results of a similar change in prison management are even more astonishing. They show a saving to the State, on account of the prisons, of $474,408.02 in the expenditures of 1878 as compared with those of 1876.

There remains the department of State charities, into which reform has not yet been introduced. That this reform should have been the last to be undertaken, and that it should promise to be the most difficult of all, is a sad commentary upon our vaunted Christian civilization. Certainly the sick, the insane, the blind, and the deaf-mute have the first and highest claim upon public sympathy and protection. And even in a financial point of view, the department of charities is more important than that of the prisons. Within the last decade, more than four millions have been expended for construction, and for repairs and improvements, upon the four hospital asylums of the State alone. This money has been asked for upon false pretenses, gained by lobbying and log-rolling, and expended by the same class of men—in many cases by the same men—who have robbed the State of hundreds of millions in the construction and repair of its canals. The asylums are the last strongholds which remain to these corrupt rings, and a desperate defense of them may well be anticipated. Several millions will be required to complete those not yet completed, and there is no limit to the sums which may be drawn from the State treasury on account of repairs and improvements to them. Besides these are the annual sums to be expended for their maintenance accounts. The current expenses of all the State charitable institutions amount to more than $1,000,000 annually, and it is expected that the joint expenses of the canals and prisons for the current year will be less than this sum.

But asylum reform has other objects than economy, essential as that undoubtedly is, both as an end and as a means to more important ends. Some of these are discussed in the comptroller's report on the State charitable institutions, and with an ability and thoroughness really surprising when the brief time permitted for their study is considered. Other writers, official and unofficial, have also contributed to the subject during the past year, and we shall take the present opportunity to give a brief resumé of the progress of this reform in the State of New York for that period.

First in order of time comes the eleventh annual report of the State Board of Charities, published in the spring of 1878. This report is important as marking the first successful effort of the Board towards practical reform in the charitable institutions of the State. The "perplexing and arduous labor" of an investigation of the State Institution for the Blind, at Batavia, was thoroughly performed, with results which may be briefly stated. The duties of its managers were found to have been entirely neglected. Its business methods were of the loosest kind, its financial accounts deceptive, and its expenditures extravagant and corrupt. As an important step in reform, the Board recommend that its administration be made a unit, by giving the entire control of its affairs to the superintendent, instead of dividing it with the steward, as heretofore. "Anything like division of authority in such an organization," say the Board, "must inevitably produce confusion and disorder." This is the theory of the administration of our State lunatic asylums. Nothing like division of authority is known in them, and this principle has been carried so far as to permit the superintendent to choose his managers, as well as all his subordinate officers. Yet the hospital asylums of New York stand convicted of enormous extravagance, deceptive accounts, and other abuses, extending over a period of years. The truth no doubt is, that where the management is entirely formal, as in this country, unity of administration is the surest means for making the commission of abuses easy, and their detection difficult. In Great Britain, on the other hand, where there is a real management of asylums by local managers, it is made the more easy and effective by a division of administrative responsibility between the medical officers, steward, and matron. But a real management of State asylums by local Boards is clearly impossible with us, and we believe, with the Comptroller, that these Boards should be abolished, and the French system adopted, in which supreme authority is given to a single officer, who is made directly responsible to one of the departments of the State government.

As we should expect, after its ten years' careful study of the various problems relating to the insane, the State Board is able to present much of interest and value in its report. Upon one point, however, it has adopted from the lunacy specialists a series of fallacies, which are a blot upon an otherwise excellent document. This is the argument in favor of the "curative policy" in providing for the public insane. The Board urge the treatment of all the acute insane in curative hospitals, lest they "lose the benefits of treatment during the period of the disease offering the greatest promise of recovery, and thus pass into the chronic stage of insanity." Thus far the argument is sound, and may be fairly used to induce that prompt removal of insane persons to the care and control of an asylum, which, in a large majority of cases, their own good and public safety demand. But this is all there is any warrant in science for claiming in behalf of the policy of early treatment in insanity. We regret to say, there is no foundation in facts, or in admitted authority, for the following statements from the report of the Board :

"Although differences of opinion exist among scientists as to the curability of insanity, it is reasonably certain from statistics upon the subject that fifty per cent. of the acute insane will recover when they are placed promptly under appropriate medical treatment. If a period of six months intervenes without such treatment the ratio of recoveries will be reduced to, and probably fall below, thirty-five per cent. The prospects of cure are greatly diminished as the disease progresses without treatment, the chances of recovery after two years' duration of the insanity being only about seven per cent."

We need hardly remark that there is no difference of opinion whatever among scientists as to the curability of insanity by medical treatment. Nothing is known of its curability by medicines, nor is any such knowledge claimed by a single authority. An articulate theory, even, of this cure has never been propounded. There is no more ground, then, for saying that "fifty per cent. of the acute insane will recover when they are placed promptly under appropriate medical treatment," than for saying that the same proportion will recover if medicines are carefully withheld from them. The cases of insanity which recover are nearly all those which cannot be connected with bodily disease, and their treatment by medicines is therefore wholly irrational. Certainly, no mechanical or chemical agent has been found capable of changing unhealthy into healthy thought and feeling, and experiments with these agents upon the insane are an abuse which should be denounced by every humane physician. It is true, indeed, that a large proportion of the insane who do not recover before the end of six months do not recover at all. But as this is at least as true of those who have not, as of those who have been treated with medicines, nothing is proved.

In a paragraph following that which we have quoted a calculation, based upon the easy curability of insanity in its early stages, is made to show a loss to the community of at least $70,000 for every one hundred insane who fail to receive medical treatment in the acute stage of their disorder. Two years are allowed for a cure in each case, at a cost of $250 per year, and against this is reckoned the cost of providing for the larger number of incurables where treatment has been neglected. Given the number of acute cases occurring in a certain period, and "the economic benefits secured to the public by proper medical treatment may readily be computed." To this the writer naively adds: "The mere statement of these facts bears its own commentary, and argument seems unnecessary to form proper conclusions regarding the matter."

It is hardly worth while to note that on the basis of the data assumed, the "economic benefits" are given at double their true amount, in this report. But that an argument so thoroughly unsound in its facts and assumptions should be found incorporated in the report of a Board made up in great part of scholars and philanthropists, is almost incredible. We can only suppose that this portion of the document was prepared by some new members whose knowledge of the subject had been wholly derived from the annual reports of our lunatic asylums.

And here we may pause to inquire, how it is that the medical writers of these reports are permitted to retain their standing in a profession whose code of ethics they so constantly and grossly violate? The argument above described is repeated year after year in nearly every asylum report in this country. Suppose these gentlemen should advertise, at their own expense and for the benefit of their private practice, that "it is reasonably certain from statistics that fifty per cent." of all acute cases of bronchitis will recover if "placed promply under appropriate medical treatment;" and that "the ratio of recoveries will be reduced to, and probably fall below, thirty-five per cent." if such treatment be delayed for six months. We know very well that fifty per cent. of the acute cases reported as cured would have been cases of a common catarrh, with a natural duration of from one to two weeks; while sixty-five per cent. and upwards of the six months' cases would have terminated in a consumption, no matter at how early a stage their treatment was begun. There is no need to say how prompt and severe would be the discipline applied to such advertisers. And yet these curers of consumption "in its early stages" are more excusable than the medical superintendents whose stock advertising argument the Board of Charities have unfortunately adopted. It has been found that certain medical agents are competent to bring about certain physical and dynamical changes in the tissues, temperature, pulse, etc., of the sick, which have been followed, at least temporarily, by improvement in the rational symptoms. How easy for the general practitioner, urged by the necessity of breadwinning, and by the eager self-deception of his patient, to deceive himself in respect to the curative effects of medicines! But, as we have seen, there is no reasonable excuse for this self-deception in the treatment of insanity in public institutions. The argument for its curability by medicines is absurd in theory, and supported in statistics only by such fallacies as we have exposed. Those who have devised it, moreover, are servants of the State, bound to their responsible duties by the highest obligations, and raised above the ordinary temptations to falsehood and deception by liberal salaries.

It is indeed a grave practical error of the Board to hold that all cases of insanity have an acute stage in which if medical treatment be given them one-half will be cured. Much more than half of those added to the number of the insane each year have never presented this acute stage, and are equally certain to prove incurable whether with or without medical treatment. Nor is there any practical difficulty in determining which these cases are. Of the yearly admissions to our asylums at least one-half are recognized as without reasonable hope of recovery at the time they are admitted, or after a few weeks' observation. This early separation of the curable and incurable insane has long been carried out in other countries, and is in fact the key to that problem of the relief of our over-crowded asylums which the Board has so long endeavored to solve. We believe they will seek in vain for its solution while they hold that "the accommodations for the acute insane are inadequate to the present requirements," and suppose this to be "fully attested by the fact that the poor-houses and county asylums give shelter to large numbers of this class for whom there is no room in our State institutions." In fact, there is ample room in the hospital asylums of New York, and of most other States, for all the acute insane, and all of the chronic class who, for various reasons, require a similar care. The first step in asylum reform should be to remove from these asylums all the cases unnecessary or improper to be maintained in them, and to forbid the admission of others of the same class. Such are now admitted and retained simply for the money which they bring to support a system of extravagant and corrupt expenditures. We hope that the report of the Comptroller has done something to bring the Board to this conclusion.

The next effort for asylum reform in New York was the result of discussions and formal resolutions by the Medico-Legal and Neurological Societies of New York City. In March, 1878, a resolution was introduced into the Legislature, ordering an investigation of the lunatic asylums of the State. Among these was specially mentioned the asylum at Utica, against which, as declared in the memorial accompanying the resolution, "grave charges had been made, by responsible and competent persons." These charges were contained in an editorial in the *N. Y. Medical Record*, of Feb. 2, 1878, which was partially based upon facts given in a review of "Bucknill's Notes on Asylums for the Insane in America," in the number of this journal for October, 1877. The editorial was an elaborate analysis of the reports of the Utica Asylum for eleven years (1866–76), and a comparison of its expenditures with those of a large number of other asylums of the highest class, throughout the United States. It afforded indisputable proofs of enormous extravagance in the expenditures of that asylum, and startling evidences of deception in its financial accounts. These charges were substantially confessed by desperate efforts to prevent a legislative investigation of them. One of these efforts was the publication in the newspapers of the report of a Committee of the State Board of Charities, directed to examine into the administration of the lunatic asylums of the State. No charges were before the committee and no witnesses or books were examined at Utica, but questions as to methods, duties of officers, and the like, were addressed to Dr. Gray, whose replies were so satisfactory as to gain the confidence of the committee in his administration "both medical and financial." There was no medical man on the committee—indeed, to its great misfortune, there has never been one on the Board—and their approval of the medical administration can have but little weight. As to the finances of the asylum, which were the sole foundation of the charges of the *Record*, the committee declare, explicitly, that "they did not go into an examination of the accounts, as that would have been a duty entirely beyond their strength and time." To urge such a report as a plea against the investigation of charges of enormous expenditures concealed by a deceptive system of keeping accounts, could hardly be deemed less than a confession. Yet for the asylum ring, as yet unscathed, in combination with the broken but still powerful canal and prison rings, this excuse proved sufficient for its purpose.

The agitation of this subject in the Legislature of 1878 was, however, the cause of a most important step in the progress of asylum reform. Near the close of the session, a law was enacted providing for an examination, by the Comptroller, into the financial and business affairs of the State charitable institutions. The main facts gathered by this examination appear in the special report of the Comptroller, but some of them are given in his annual report, published at the beginning of the year. In this he renews, with additional emphasis, the recommendation contained in his report of last year, "that the receipts of our charitable institutions be covered into the treasury of the State, and that an appropriation sufficiently large be made for their maintenance." After describing a case of outrageous extravagance, accidentally detected in one of the State asylums, the Comptroller says:

"Another case deserving criticism is brought to light in one of the State institutions, which publishes a *Journal of Insanity*. This paper has been published for many years, and from an account rendered to this department, at my request, I learn that since 1850 its bills have been paid out of the funds of the institution to the amount of $17,092.42. Up to 1856 the institution received from the paper, $670.53. Since 1856, or for twenty-two years, the treasurer reports no receipts from the paper, but says: 'I have understood and believe that they have been spent in editorial services, and the purchase of books for the asylum libraries.' It would seem then that one at least of our charitable institutions not only has large miscellaneous receipts, the vouchers for the expenditure of which never come to this department, but that it has receipts the amount of which its own treasurer cannot state, and the vouchers to account for which he never sees. It would be difficult to properly stigmatize such loose management."

In another place, the Comptroller renews a recommendation made in his report for the previous year, as follows :

"I would recommend for your consideration the policy of abolishing all local boards of trustees, and the erection of a system by which the different institutions shall be managed by one controlling power. As it is, the responsibility for loose and expensive management is not centered in any one. If we are to look for improvement it can only be found in establishing a fixed and definite responsibility in place of the present plan where there is, practically, no accountability."

A resolution embodying these views was introduced into the Senate during the late session, but was tabled on the motion of the senator from the Utica district, and failed to become a law. According to custom, this senator selects the three managers of the Asylum nominated by the Governor to the Senate each year. As stepping-stones for aspiring politicians, and as shelves for decayed ones, as well as favorable points for gaining access to the State treasury, these managerships are much valued. Thus the asylum boards are made up of local politicians, by whom the interests of the State and the insane are as little considered as abstract justice by an advocate in a legal contest. The appointment of politicians from both parties to these boards, instead of making them non-partisan, as was intended has made them doubly partisan, and almost impregnable. Excellent as the recommendations of the Comptroller seem to be, they have little chance of being adopted at present.

The special report of the Comptroller upon the charitable institutions of the State, whatever may be its immediate effects, is certain, we think, to mark an epoch in the history of the State asylums. Their financial and other statistics, from the beginning, are tabulated with such admirable method and accuracy as to furnish complete data for investigation and reform. With what infinite labor this task has been accomplished, the readers of the JOURNAL who have followed its exposures of the deceptions, concealments, and juggling with figures of the Utica asylum, will partly appreciate. We shall only say, that the report presents in a light more striking than they have yet been seen, the marvelous extravagance of the hospital asylums. Lack of space forbids us to follow it in detail, but it is only just to our readers to copy the following contrast of the financial administration of the asylum at Northampton, Mass., with that at Utica, in proof of the correctness of our criticisms of the latter :

"It appears from the report of the Superintendent of the

Utica Asylum that for the year ending November 30th, 1878, there was received from sundry counties for patients' board, clothing, and miscellaneous expenses, the sum of $101,946.51; from sundry private patients, $54,805.57, making a total of $156,752.08 received from counties and private patients. Dividing this sum by 600, the average number of patients for the year, we find that the average amount received for each patient was $261.25, or a trifle over five dollars per week, as against three dollars seventy cents and eight mills at Northampton. To this latter sum, however, should be added a few cents, not to exceed twenty, to cover the amount received for clothing and damages from town and private patients. It therefore appears that for the year 1878 the amount received on account of patients was at least a dollar and ten cents per capita per week more at Utica than at Northampton; or stated for the year, the amount received at Utica from counties and private patients was $57.25 per capita more than was received from corresponding sources at Northampton. The excess of receipts from these sources at Utica over Northampton was greater, rather than less than this amount, for the twelve years preceding 1878, yet we find that while at Northampton during the years 1866 to 1878 inclusive, every expense, including repairs and improvements to the extent of $156,701.31, has been met out of the receipts from patients, the Utica Asylum, with its receipts from this source $57.25 per capita for the year greater than at Northampton, has received from the State, during the thirteen years from 1866 to 1878 inclusive, the same period covered by the figures given above, $156,159.37 for salaries and $384,886.71 for additions, alterations, repairs and other purposes, making a total of $541,046.08 received from the State, in addition to the amount received on account of board of patients. This contrast appears the more striking, when it is reflected that the excess of receipts from patients, $57.25 per capita multiplied by the average number of inmates in the Utica Asylum for 1878, produces the sum of $34,350 for that single year."

When to this excess of $34,350 in the receipts of the Utica asylum for maintenance in a single year is added the further average sum of $41,619 received from the State treasury for salaries and for repairs and improvements, the total ($75,969) is so large that without the fullest explanation it will be classed with the sums expended on account of the canals and prisons of the State before the days of reform. The entire excess, for maintenance and for repairs, etc., in the Utica expenditures, has amounted to a little more than a million of dollars in the past fifteen years. It cannot be the true friends of the State charities, or even of the Utica asylum, who combine to stifle inquiry into such an accusation as is contained in these figures.

For its permanent value as a reference, we shall also copy from this report a tabular statement of the total cost of support per patient in each of twenty asylums of the highest class, for the year 1877. It will be seen that in the asylums at Poughkeepsie and Middletown, the cost is even greater than at Utica. But neither of these asylums is completed, and their expenditures are a less perfect index of their management than those of the parent institution. The table shows that the total cost per patient at Utica for 1877, was almost exactly double that at Northampton. This is a difference of $110,000 against the expenditures of the former asylum for the year. A comparison of the maintenance accounts of the two institutions shows an excess of $71,744 in those of the Utica asylum for an equal number of patients, and when we consider that at Northampton the cost of the principal supplies is from five to ten per cent. greater than at Utica, the difference is relatively increased.

NAME OF INSTITUTION.	Average number of patients.	Annual cost per capita excluding repairs.	Annual cost per capita including repairs.
1. Asylum for the Insane, Toronto, Ontario	651	$128 30	$133 39
2. Asylum for the Insane, Brattleboro, Vermont	472	136 07	150 77
3. Asylum for the Insane, London, Ontario	604	136 84	140 20
4. Hospital for the Insane, Halifax, Nova Scotia	347	140 46	154 27
5. State Lunatic Asylum, Jackson, Mississippi	350	161 34	167 58
6. Hospital for the Insane, Dayton, Ohio	571	161 88	166 00
7. State Lunatic Hospital, Taunton, Mass.	727	162 81	194 90
8. State Lunatic Hospital, Northampton, Mass.	476	166 34	183 72
9. Hospital for the Insane, Cleveland, Ohio	577	170 35	177 19
10. Hospital for the Insane, St. Peters, Minn.	563	177 77	180 60
11. Central Insane Aslum, Jacksonville, Ill.	487	185 52	217 81
12. Government Hospital for the Insane, Washington, D. C.	770	202 69	213 64
13. Hospital for the Insane, Oshkosh, Wis.	542	206 11	216 92
14. Maine Insane Hospital, Augusta, Me.	411	208 49	219 45
15. State Lunatic Asylum, Harrisburg, Pa.	434.	213 93	231 59
16. Hospital for the Insane, Middletown, Conn.	463	226 24	240 34
17. Hospital for the Insane, Catonsville, Md.	245	235 15	246 01
18. State Lunatic Asylum, Utica, N. Y.	608	283 96	362 53
19. Hudson River State Hospital, Poughkeepsie, N. Y.	219	353 20	365 59
20. State Homœopathic Asylum, Middletown, N. Y.	109	419 75	431 14

The argument for the "curative policy" in the care of the insane, which we have noticed in the report of the State Board of Charities, the Comptroller terms "an ingenious argument for high-priced management," and thoroughly demolishes. He shows that, in New York, "since 1850, during the very period when the cost of asylum management has so largely increased, there has been a steady decrease in the reported recoveries." After quoting on this point from a "Report on Insanity," read before the Conference of Public Charities in 1877, he says:

"Dr. Chapin's deductions are more than confirmed by further investigation. The Utica Asylum was opened in 1843. The percentage of recoveries for the first five years of its existence, based on its average population, was 49½. The percentage of recoveries on the average population for the last five years has been 22½. The difference is not so striking when the comparison is made of the percentage of recoveries basedon admissions, but in this case also the percentage is much smaller for the last five years than for the first five years in the life of the institution. On the other hand, the percentage of deaths, whether based on the whole number treated, or on the average population, has been larger for the past five years than for the first five years after the opening of the asylum, yet, in the intervening time the expenses have doubled."

Dr. Earle's able "Studies relative to the Curability of Insanity," published in his last annual report, are quoted in confirmation of these deductions. This veteran inquirer finds that the McLean Asylum, at Somerville, Mass., with an immense increase "in the elegancies of accommodation, and a nearly sevenfold augmentation of current expenses," reports a ratio of recoveries diminished sixty-one per cent. between the years 1840 and 1875. This really proves nothing so much as that the cases now placed in asylums differ very greatly from those of former years. But at least it "effectually disposes of the argument for a high rate of expenditure, based on the theory of increased curative results," as the Comptroller believes.

The misleading financial statements which appear, year after year, in the reports of the Utica Asylum, and especially in its returns to the State Board of Charities, are treated by the Comptroller with a fine irony which is very effective. He says:

"It is not a necessary inference that there existed an intention to mislead. The trouble is as likely to have been that the person making the report, or furnishing the figures, *failed to bear in mind the object of publishing them.* Yet it may be that, in some cases, a desire to avoid an appearance of extravagance has led to such a purposed arrangement of figures as should not enable the general reader to gain a full knowledge of the subject."

We have before shown* how conscious and deliberate are these deceptive arrangements of figures in the accounts of the asylum referred to, and that they have amounted at times to $50,000 in the statement of its weekly cost per patient for a single year. And we may observe here that the only explanation which has been offered, within our knowledge, of its enormous expenditures, is destroyed by this report. It has been suggested that a medical and philanthropic fanaticism for the insane inspires the administration of the Utica Asylum. But the Comptroller finds, by a careful examination, that this extravagance does not reach the patients. Besides, if the suggested explanation were the true one, we should find the representatives of the Asylum glorying in their extravagance, and not concealing it by false returns, or manipulating Legislatures to prevent an investigation.

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* Vol. II., page 782.

It would be a pleasure to add the thoroughly fair and sensible remarks, with which this valuable report concludes, on the necessity of economy in the use of money for charitable purposes, and especially in the cost of asylum buildings. But we must hasten to the latest incident in the history of asylum reform in New York.

On the 8th of May last, several well-known medical gentlemen of New York City appeared, according to invitation, before a committee of the Senate at Albany, to give the facts and reasons upon which had been based a petition for the investigation of the State insane asylums. That petition was published in the last number of the JOURNAL, with the comment that the statements made in it with respect to the management, medical, financial and administrative, of these asylums, were serious, and that the fullest examination of them should be made. This opinion was only partially concurred in by the superintendents. They deemed the charges serious enough, as it would seem, but were strongly opposed to an investigation. This view they were permitted to present to the committee in a formal hearing, before the charges against them were heard, and with complete success. The committee, whose leading member was the senator from the Utica district, reported against the petitioners, and the dreaded inquiry was once more avoided.

The first series of charges were directed to the administration of the asylums. They accuse the superintendents of gross neglect of duties which they are liberally paid by the State to perform, and of some serious abuses in the treatment of the insane. In no way could a confession of the general truth of these charges have been more clearly made, than by the efforts put forth to prevent their investigation. But it would be wrong to suppose that all the superintendents were equally guilty, or that there were none who were quite guiltless of these offences. Some, we are sure, were moved by an *esprit de corps* arising from fellow-membership in a narrow and exclusive society, still governed by the spirit and traditions of the last century. We believe that these gentlemen have done a serious wrong to the public and to the medical profession not only, but to themselves.

Their absolute and irresponsible control of asylums is already lost, and no attempts to shield their methods from public and professional criticism, or their unworthy members from reproach, can long avail. Of the truth of this the better class of asylum superintendents, who are largely in the majority, should be assured.

The second series of charges relate especially to the professional qualifications of the superintendents. That not any of them are qualified according to the standard of the petitioners, is no doubt true, and it is quite impossible that it should be otherwise, under the present organization of asylums. Each superintendent is now practically the steward of his institution more than its physician, and to these functions, as the Comptroller has pointed out, is unfortunately added that of lobbyist. In our opinion he is in general much less blameworthy for what he has not done in pathological studies, than for making a pretense of them for advertising and lobbying purposes. This offense is plainly charged, and the proofs of the charge are so clear that no medical reader of asylum reports can fail to be convinced. We have before referred* to Dr. Gray's summary of contributions to the pathology of insanity, after ten years of study, at an annual cost to the State of $5,000 and upwards. "It is impossible," says Dr. Gray, in opening, "to define the pathology of insanity in distinct terms, as it is still a subject of investigation." Can we hesitate for a moment as to the real value of studies undertaken from such a scientific and logical stand-point? Would it be possible to imagine Dr. Draper or Dr. Dalton at a loss to define any province of physiology, and for the reason that it is still a subject of investigation? What province of physiology or pathology is not still in this state, and what has this fact to do with the task of defining it? We ask the reader's pardon for dwelling an instant on so obvious a point, but it furnishes a true key to the entire field of Dr. Gray's "researches," which have filled from twenty to forty pages of each of his annual reports for many years. They consist of a description of degenerative changes found in the brains of the insane, which have long ago been proved to have no definite relation to mental disorder. Thus they are as far from having any value in the pathology of insanity as a description of the same changes in the brains of inebriates—where they are also found—would have in a pathology of inebriety.

But it is not necessary to dwell longer upon so unpleasant a subject. We are aware that it will be admitted by those even who consider themselves the special friends of asylums and the insane, that these so-called "studies" are not really serious, but intended solely for popular and legislative reading. There can be no doubt of this fact in the mind of any physician who has been fairly grounded in the principles of medical science. But we must again warn medical superintendents that the public mind, at the present day, can be only temporarily controlled by "arguments" of this sort. Its reasoning, though but partly conscious and articulate, finally leads to just, practical conclusions. Enlightened public opinion, indeed, is the only ground of real and permanent reform; and when it shall have become fully understood that such delicate and responsible public duties as the care of the insane are committed to officials exercising absolute authority, accountable to no one for their expenditures, and openly combined to resist all attempts at investigation, asylum reform will be no longer delayed.

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* Vol. III., page 352.

In conclusion, we shall present a resumé of the changes in asylum organization and management, suggested by the movement for reform now in progress in New York, and by our own observation and experience:

1. To abandon the theory of asylums which considers them chiefly as lunacy-cures, established for the benefit of local interests, and to be maintained by lobbying and advertising.
2. To abolish the local boards of trustees, and adopt a system of State management for State institutions, with a single responsible head, in place of the present formal management.
3. To devolve the medical and general supervision of asylums upon the State Board of Charities, which should be composed in large part of physicians.
4. To enact a law similar to that providing for the removal of trustees of savings banks for abuses of trust, so that commissioners or trustees, charged with the supervision of asylums, and neglecting or abusing their trusts, might be summarily removed.
5. The enforcement of a law never carried into effect, authorizing the admission of private patients into asylums only when there are vacant places not required for public patients; and the charging of a uniform price of maintenances to both these classes, instead of leaving, as now, the price to be paid by private patients at the discretion of the superintendent. L. A. T.

II.—DURET: CEREBRAL TRAUMATISMS, &c.

Études Expérimentales et Cliniques sur les Traumatismes Cérébraux. Par le Dr. H. Duret. Paris, 1878. Pp. 369. (*Experimental and clinical studies of cerebral traumatisms.*)

(*Continued from January No.*)

In explaining the temporary tetanic symptoms which sometimes follow a severe blow on the head of an animal, some interesting remarks are made, which may have a wider range of application than the author seems to have given them. At any rate we will place them before the reader. He says, "The tetanic state extends alike to the muscles of the vegetative, as well as those of the life of relation: 1st, the muscles of the members, of the face, of the trunk, of the tail, the eyes, &c., pupillary contractions, muscles of respiration, &c. ; 2d, muscles of the rectum, of the bladder, of the salivary and lachrymal ducts, &c., and vascular muscles. There is not the least doubt, but that a 'contracture' so rapid, so general, and having such intensity, is reflex. What is the sensitive part, which is, or may be the point of departure, of the irritation provoking the reflex? It is the medulla, and in the medulla, is chiefly the *restiform* bodies, which are so much injured by the shock of the cephalo-rachidian fluid," produced as was shown in the earlier part of this notice. The author proceeds to support his opinion, that the restiform bodies are chiefly involved in the production of the tetanic phenomena, by the following arguments:

That they are found to be the especial seats of injury in the examination *post mortem* of such cases. That the "restiform bodies are sensitive parts, and mechanical irritation of the same, by experimental means, produces the same tetanic phenomena," as have been seen in Dr. Duret's cases. Even very slight mechanical irritation of these bodies produces a general tetanic movement, its severity being proportioned to that of the irritation, at times producing complete opisthotonos. At this point Dr. Duret supports himself by the authority of his former master, Professor Vulpian. Dr. Duret declares, that irritation of no other part of the medulla produces such phenomena with so much readiness and perfection. Though not a new fact, yet this is an important one, one upon which enough stress has not been laid by clinicists in their endeavors to localize centrally such disorders. Inasmuch as this is an important point, we will quote at greater length from our author: He says, "In no other part of the nervous system do we find, except perhaps in the peduncles, and the posterior part of the internal capsule, collected so many sensory fibres, which by reason of their short course, exert a powerful action on the reflex motor centres, with which they stand united. Why not admit then, that the violent reflex tetanic contractions are the result of their excitation?" In the course of this discussion, Dr. Duret denies that the phenomena could be due to irritation of the dura mater, or its nerves, as there exist reasons for thinking may be true, at least in some cases. The extreme vascular perturbations, which occur in cases like those under discussion, are examined at some length, but nothing of importance to our readers is developed, since this class of subjects has been discussed in the Journal, many times at great length, from its first appearance.

But it may not be amiss to note at this point that during the presence of the tetanic symptoms, Dr. Duret believes himself to have demonstrated that there exists a spasm of the whole arterial system, of the brain as well as of the periphery, and that with a disappearance of the tetanic symptoms, there is a contrary state of the vessels.

But we would not pass on without calling attention to the significance of the fact, as it seems to be, that the restiform bodies are sensitive. The fibres of which these bodies are composed appear to go, at least mostly, to the cerebellum. Do any considerable number among them go elsewhere, or is the cerebellum sensitive? This is a question that has never been satisfactorily answered, so far as we know, but into a discussion of which we cannot now enter, and in relation to which, in the seeming conflict between anatomical and physiological research, we have not been able to arrive at definite opinions.

In drawing practical conclusions from his experiments, consisting of blows on the head, of varying degrees of force, and of observations of the phenomena which resulted, Dr. Duret finds that blows on the *front* of the head are more grave than those made on the lateral regions of the same.

Among the other experimental cases detailed of great interest, is one of a sudden traumatic locomotor ataxia, the principal lesion being in the posterior columns of the upper portion of the spinal cord.

After a prolonged study of the phenomena belonging to each variety of shock, Dr. Duret deduces the following general laws:

1. "When a localized lesion is produced by the traumatic action of the cephalo-rachidian liquid, in any region whatever of the myelencephalon, it is revealed by certain signs, which are in relation with the functions of the wounded part.

2. "In the first period of the phenomena of shock, the signs differ according to the degree of intensity of the lesion produced.

(1.) "If the destruction of the wounded part (centre or conductor) is complete, there is a loss of function, a paralysis.

(2.) "If the lesion is light and non-destructive, and consists in a light shock, there is or will be, by mechanical irritation, exaltation of function.

3. "In the second, or period of congestive and inflammatory reaction, if the destruction of the part is complete, we may nevertheless observe at first phenomena of exaltation generally, finally succeeded by paralysis.

4. "In all periods there may be exhibited *phenomena of diffusion* of symptoms, such as epileptiform attacks, contractures, reflexes, &c." (P. 137.)

But it will not be possible to refer to all the suggestive results arrived at and comments made by Dr. Duret in this portion of his memoir. We strongly commend them to our readers—especially the summary found between pages 153 and 157.

Next follows a discussion of intra-cranial pressure, in which the researches of Leyden and others are mentioned, and their results substantially adopted, to the effect, that there exists a degree of intra-cranial pressure which cannot be referred to the atmosphere. M. Duret in the experimental treatment of this subject, employs mostly localized pressure, such as results where wax is injected between the dura mater and bony walls of the cranium, or between the dura and brain, the injection being made through a small hole in the cranial wall. The practical conclusion reached is that such localized compression exerted on the surface of the hemispheres causes general anæmia of the brain and medulla, thus causing in various degrees cessation of the blood-supply to the same. What happens in such experimental lesions, no doubt happens in the accidents to which human beings are subject, in which there is fracture with depression, or meningeal or other hemorrhages. The explanation of the phenomena in the one case, is applicable to those of the other.

This subject of intra-cranio-spinal pressure, we regard as one of extreme importance, not only in relation to such researches as those of Dr. Duret, and the recent researches of Leyden,* but in relation to changes in barometrical pressure, and the widespreading effects of the same, on intra-cranial pressure, the relations of heat and cold to the same, on account of changes in the cutaneous circulation, and corresponding thereto, changes in intra-cranial vascular pressure, and also changes in posture and its effects on the same. These subjects have not been studied as they deserve to be, and in the future will be, in their hygienic and therapeutic relations, but we cannot enter on a consideration of the same now. There is an interesting chapter, on intra-ventricular hemorrhages, especially in relation to the muscular "contractures" which have been so often observed to follow them. Dr. Duret has produced experimentally, the same lesions as have been observed clinically by Charcot, Vulpian and others, and with essentially the same results. The "contractures" produced by intra-ventricular hemorrhages are due, according to M. Duret, directly to "irritation of the peduncular expansion, or of the restiform bodies, that is to say, of the sensitive parts neighboring to the seat of hemorrhagic lesion."

But this explanation by M. Duret of the mechanism of the "contractures" mentioned, following hemorrhages into the ventricular cavities, has been lately contested by M. Cossy, in a series of observations which it is our intention to give the results of in a future number of the Journal, and which were referred to briefly in our last issue (P. 370). He denies that the cause of the "contractures," is due to irritation of the sensory parts of the peduncular expansion, more especially of the restiform bodies. M. Cossy found injections into the lateral and third ventricles, which did not appear to involve the restiform bodies, to produce the "contractures." He concludes they must be due to irritation of the excito-motor parts of the internal capsule and cerebral peduncles. He moreover denies, that the convulsions due to blows on the head are due to shock in the fourth ventricle, produced by the cephalo-rachidian fluid, as claimed by Dr. Duret. He found that convulsions due to ventricular hemorrhage did not follow any more certainly if in the fourth, than if in the lateral ventricles. Indeed, in twenty cases of hemorrhage in which blood reached the fourth ventricle, in eight only were convulsions present, in the other twelve they were absent. But we do not see that M. Cossy's observations invalidate the fundamental positions assumed by Dr. Duret, or that the latter has correctly explained the mechanism of his own cases.

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* *Archiv fuer Psychiatrie u. Nervenkrank.* Band IX.

We now come to the most valuable portion of Dr. Duret's work, at least on some accounts. It relates to the part played by the dura mater and its nerves in case of injury and disease. The profession, so it seems to us, is only beginning to be aware of the great importance of this membrane in intra-cranial disease.

This part of the work is very properly introduced by an account of the nerves of the dura, which, as it should be remembered, are chiefly from the fifth pair. Those fibres which supply the ethmoidal and orbital portions of the dura, are chiefly from the ethmoidal branch of the nasal, which is from the first division of the nerve. Those supplying the middle region come from the ganglion of Gasser, according to Cruveilhier, those supplying the posterior come from the ophthalmic branch of Willis of the fifth. The nerves of the dura lie quite near its free surface, and are quite abundant. This membrane is not only highly sensitive, but is very vascular. The dura mater is exquisitely sensitive, especially in disease, as is well known.

At this point our author begins the recital of experiments on this membrane, by which he is able to show indubitably, that by irritation of the same produced in various ways, reflex movements were readily produced not only in the voluntary muscles of the face, members, eyes, &c., but of the involuntary, as those of the iris, heart, vessels, &c.

Dr. Duret carefully distinguishes, as some others have not done, between the reflex movements produced by excitation of the nerves of the dura, and those which result from irritation of the cortex of the brain, now so well known since the original researches of Hitzig, Ferrier and others.

Dr. Duret partly agrees with Dr. Dalton, of New York, that the convulsions which result from irritation of the dura, are more likely to be on the same side as the irritation, than is the case with convulsions caused by so-called irritation of the cortex; in this latter case the convulsions are always, according to Dr. Duret, on the opposite side to that operated on. In this part of his subject he quotes to considerable extent the researches of his collaborator, M. Bochefontaine. If the irritation of the dura was mild, the convulsions were restricted and were usually on the side operated on, as will be readily understood by those acquainted with the mechanism and modes of action of the nervous system.

If the irritation is more severe, then the convulsions were not only more wide-spreading, but often extended to the opposite side of the body, as may be readily understood when we consider the structure of the medulla, and the wide range of central relations of the trigeminal root in the same. In cases of irritation of the dura, which lead to reflex vascular disturbances through the vaso-motor nervous system, it is noted by Dr. Duret, that the cerebral circulation is especially liable to derangements. This we regard as an exceedingly important fact, in its clinical relations. For what is observed in experimental cases, is also true for pathological clinical cases. It is exceedingly common to have localized disease of the dura which may and does give rise to a multitude of symptoms according to its extent and seat, as well as its nature and degree of acuteness. Dr. Duret feels authorized to declare as the result of his observations, that "contractures," secondary to lesions of the convexity of the hemispheres, are due to irritation of the nerves of the dura mater. He generalizes as follows: "Lesions of motor regions, centres or conductors, manifest themselves by localized clonic spasms, by spasms generalized under the form of epileptic attacks, or by paralyses. Lesions of sensitive fibres are revealed by pain, by hyperæsthesias, by anæsthesias, or by *reflex motor phenomena*, 'contractures,' or tetanic attacks." And according to our author, the chief seat of the sensory irritation which produces such phenomena (so far as the cranium is concerned), is the dura.

The work then concludes with chapters on the rôle of the vessels of the meninges in cerebral traumatisms, and on the rôle of the different parts of the encephalon in cerebral traumatisms. But under these latter heads there is at this date much less that is new or suggestive. Dr. Duret, as is known long since to our readers, accepts like the majority of French physiologists, the localization of functions in the brain, more particularly its cortex, and towards the establishment of this doctrine our author has made no insignificant contributions from his own researches. The volume is closed by quite a number of finely executed colored drawings, illustrating in an instructive manner the descriptions in the text.

Altogether this is a highly valuable and suggestive contribution to the physiology and pathology of the central nervous system. But the work was evidently prepared for the press in some haste, and in no small measure lacks in that thoroughness and strength of discussion which prevails in the works of his former masters, Charcot and Vulpian. We would not close, however, without expressing a lively sense of obligation to its author, for the benefits obtained in the perusal of his excellent work, which we do not hesitate to recommend to our readers.

III.—THE VAGUS NERVE.

I.—DIE TROPHISCHEN BEZIEHUNGEN DER NERVI VAGI ZUM HERZMUSKEL. Von Dr. Hermann Eichhorst. Berlin: 1879. A. Hirschwald. Pp. 38. (*The trophic relations of the vagi nerves to the heart muscle.*)

II.—FOLGEN DER VAGUS-DURCHSCHNEIDUNG BEI VŒGELN. (*Consequences of Section of the vagus in birds.*) Richard Zander. *Pflüger's Archiv für Physiologie*, Bd. 19, p. 263 (1879).

I. Up to the present day it is still an open question, in what manner death, the inevitable consequence, is caused by section of the pneumogastric nerves. The autopsy of the animals reveals almost always an inflammation of the lungs. But since we know, from the researches of Traube and others, that this condition is caused by the entrance of saliva, food, etc., into the insensible bronchial tubes, we can prevent it altogether by proper management. Nevertheless, the animals operated upon cannot escape death. As the true cause of death, Eichhorst claims an impaired nutrition of the heart, and bases this view upon the following observation, commencing with birds, but studying also the phenomena in mammals.

It has been claimed by Cl. Bernard that irritation of the vagus *in birds* does *not* arrest the heart. While some other observers had previously arrived at other results, the matter was particularly investigated by Einbrodt (*Müller's Archiv*, 1859, p. 439), who *did* prove an inhibitory influence of the vagus upon the heart of the bird, but found the nerve easily exhaustible. This result Eichhorst confirms fully, but he disagrees with the former author as to the cause of death. Einbrodt stated that his animals died from impaired digestion and nutrition, while Eichhorst claims that these conditions can be entirely prevented by proper care. But nowhere in the brochure does he tell us in what the care consists.

The consequences of division of the two vagi in pigeons, ravens and jackdaws are the following: The pulse is at once accelerated, and respiration becomes dyspnœic. Inspiration is deep and slow, while the expiratory effort is rather sudden, and accompanied by a squeaking sound. The respiratory movements become reduced to 20, 10, or even 5 per minute. The pulse becomes too rapid to be counted, amounting to more than 250 per minute, and the cardiac sounds lose their clear timbre and become muffled. Various other symptoms occur, but they are neither constant nor important. Attempts at swallowing and oozing of a viscid fluid from the mouth are often observed for a short time after the operation. *Contrary to all experience upon mammals, Eichhorst claims to have found no impairment of digestion in birds.* He claims to have seen them eat, and found their stomach and intestines filled—food undergoing digestion.

The disturbances of circulation and respiration diminish gradually during the course of the second day after the operation. Still, the animals are evidently sick. Their eyes appear dull, their plumage is disordered, the birds are crouched in a corner of the cage in an apathetic manner. But in a few days they regain their former liveliness, and present no appearance of suffering. Nevertheless, they die inevitably, usually about the end of the first week. The death occurs sometimes quite suddenly; in other instances it is preceded by dyspnœa for some hours.

After death no change can often be detected with the unaided eye. The author denies directly that the birds presented any appearances of starvation. *In birds pneumonia does not occur as the consequence of section of the vagi.* Occasionally a slight quantity of a clear, frothy fluid can be pressed out of the pulmonary tissue, but there is never any evidence of inflammation. This is in accordance with Traube's theory of the origin of the pneumonia from swallowing the buccal fluid and food, since this cannot occur in birds.

The heart is always found diseased after death. Sometimes no morbid change can be seen with the unaided eye; in other cases the organ is pale, relaxed and friable; most frequently it is studded with yellowish streaks and spots of varying extent—mostly near the inner surface. The left ventricle is always more implicated than the rest of the heart. The extent of the change does not depend alone upon the time after the operation, but varies in different animals. The microscope reveals always that a large number of the muscular fibres of the heart are filled with fat granules and drops, and that the striation has disappeared in the entire fibre, or at least a portion of it. The muscle nuclei can no longer be recognized.

This fatty degeneration might be referred to three conditions: the dyspnœa, the pulse acceleration, or the division of any trophic nerve fibres. The first ground, suggested by the recent researches of Fränkel on fatty degeneration as the consequence of reduced supply of oxygen, is really untenable. Previous observers have found that (at least, in rabbits), the absolute quantities of O and CO_2 remain the same as before section of the pneumogastric nerves, the disturbance of respiration referring only to the rhythm. For want of apparatus, the author could not verify this in the case of birds; but he refers at least to the perfect return of normal respiratory rhythm in the course of the second day.

The degeneration can also not be attributed to the increased work of the heart. In order to exclude this as the cause Eichhorst supplied birds during a period of even ten days with sufficient atropin to paralyze the inhibitory fibres and raise the pulse to 300 to 400 per minute. Nevertheless no degeneration of the heart muscle was produced. Hence the author suggests the existence of trophic fibres in the trunk of the vagus as the real cause.

In rabbits the cardiac changes are not seen ordinarily on account of the early death of the animals. They are usually carried off within the first twelve hours by a pneumonia caused by the entrance of food and fluid into the lungs. But the pulmonary process can be prevented by tying a canula into the opened trachea, and in this case the rabbits can survive two or four days. Taking these precautions Eichhorst could always find cardiac degeneration. The heart was soft, pale and friable, and as in the case of the birds, marked with irregular fine, yellow streaks. Many or most fibres had lost their striation, and presented the appearance of *waxy* degeneration. In birds, on the other hand, only *fatty* degeneration could be seen, there was no proliferation of nuclei, or any other evidence of an inflammatory process.

In these animals Eichhorst could also satisfy himself, that the lesion was not due to overwork, since a pulse-acceleration produced by the administration of atropin for a fortnight had no such result. The inhibitory fibres are all derived from the spinal accessory nerve. Destruction of this nerve accelerates hence the pulse as much as section of the vagi, but does not cause any cardiac lesion.

The same results were obtained in dogs. The cardiac degeneration in these animals is rather of the fatty type, but remarkably slight in extent, even if the death is postponed by the avoidance of pulmonary troubles. The difference in the intensity of the degenerative process in different animals, Eichhorst attributes to the different structure of the cardiac muscle. In dogs the fibres of the heart muscle are tough and relatively thick, while in birds they represent much more delicate structures.

The main conclusions which Eichhorst bases on his experiments are: *That the vagus contains fibres, the division of which leads to fatty degeneration of the heart-muscle, and that this condition is the cause of death.*

There is, however, nothing convincing in the author's reasoning. There is no proof whatever, except by a questionable exclusion, that death is really due to this state of the heart. No exact account of the mode of death, no graphic curves of the failing circulation can be found in the book. The author denies that the animals die of starvation, and this indeed seems improbable on account of the very short time between the operation and the fatal issue. But may not the failure of general nutrition, which all other observers *have* found, cause the cardiac degeneration, especially if we take into account the overwork of the heart? At any rate both Manasseïn and Falk have found the cardiac degeneration in starved animals. The existence of true trophic fibres in the vagus is only a *possible* or even *plausible* explanation of the author's observations, but that it is the *true* explanation he fails to prove. His entire work, valuable as it is, suggests rather new problems, than positive answers to previous ones. Eichhorst's results have induced Wassilijew to take up the same subject in Botkin's laboratory in St. Petersburg. (*St. Petersburger med. Wochenschrift*, Nos. 7 and 17, 1879.)

He found likewise a cardiac degeneration after section of the vagi in the rabbit, but attributes it to the starving condition. He obtained also the same lesions by inducing an irritative process in the nerve by pricking it. But in reality he induced by this supposed "irritation" an atrophy of many fibres of the vagus, as is evident from his microscopic examination. Hence, his conclusion as to the causation of the cardiac lesion by a continued state of irritation of the vagus, is not tenable.

II. The article of Zander impresses the reader at once as the thorough work of a good observer. Contrary to the results of previous observers, including Eichhorst, he found that section of the two vagus nerves in birds does not leave the lungs intact. In every instance the operation was followed by disseminated spots of hyperæmia and œdema of the lungs. In mammals on the other hand the nerve section causes a true pneumonia, which is mainly or wholly due to the entrance of fluid and food into the trachea. In birds this does not occur, since their upper larynx is supplied with sensibility and motility by the *superior* laryngeal nerve, which is not affected by the division of the vagus in the neck. But on dividing these nerves also, the contents of the gizzard, putrid on account of the paralysis of that organ, enter the larynx, and the result is a septic pneumonia. The same can be produced in birds in which the vagi alone have been divided, on injecting artificially such putrid fluid into the lungs.

The pulmonary hyperæmia following nerve section is more considerable immediately after the operation than a few days later. Hence, Zander considered it likely that this was due to an irritation of vaso-dilator nerves contained in the vagus, by the act of division. On laying bare the lung he did actually find hyperæmia of the lung on dividing the vagus, which gradually diminished, but could be reproduced by irritating the distal nerve end. Corresponding observations were made on the temperature of the lungs, as measured by means of thermo-electric needles. Division or irritation of the vagus increases the temperature of the corresponding lung for some hours. If both nerves are divided, the temperature in the rectum sinks a few degrees—no doubt on account of the greater loss of heat from pulmonary hyperæmia—but within some hours it rises again to nearly the normal height, and remains constant until death. If the contents of the gizzard are prevented from entering the lungs, the pulmonary changes are too slight to cause death. What is, hence, the real cause of the death?

Young birds die mostly from suffocation. Their larynx is not yet ossified, and becomes compressed, like a valve during inspiration, on account of the paralysis of the dilating muscles.

In other cases death occurred quite rapidly, without any preceding symptoms, and Zander attributes it, hence, to sudden paralysis of the heart; but this explanation is not satisfactory.

In adult animals it is even more difficult to find the real cause of death. In some instances Zander found a fatty degeneration of the heart. In other cases the post-mortem appearances are detailed, without any mention whatever of this condition. It seems that Zander did not use the microscope to any great extent. He admits, however, that this degeneration may cause death by cardiac paralysis. Nevertheless, the heart thus degenerated was found beating in some birds after failure of the respiration! The existence of trophic nerves, however, the author denies altogether, attributing this fatty degeneration to the state of inanition caused by vagus-section. At any rate, he claims to have found the same morbid process in the hearts of starving pigeons. The most important cause of death Zander seeks in the starvation due to paralysis of the gizzard and œsophagus. Although the animals can eat, food is of no use to them, since it is all retained in the gizzard, and putrifies in its cavity. It cannot even be pushed by force into the muscular stomach. The animals dying about the end of the first week presented the usual post-mortem appearances of death from starvation. Some attempts made by the author to maintain life by artificial feeding did not succeed.

Notwithstanding the care betrayed in the paper, as well as the many new facts found in it, the cause of death after vagus-section is not quite clear. As it appears from the accounts of his experiments, the animals died often before they had lost forty per cent. of their weight, a loss found regularly by Chossat. They died, besides, within a period of time rather too short to permit death by starvation. H. G.

IV.—RICHET: CEREBRAL CONVOLUTIONS.

Structure des Circonvolutions Cerebrales (Anatomie et Physiologie). These presentee et soutenue a la Faculte de Medicine de Paris pour le Concours de l'agregation (Section d' Anatomie et de Physiologie). Par le Dr. Charles Richet. (*Structure of the Cerebral Convolutions.*) Paris, 1878. 172 pages. 2 litho. plates.

The first part of this memoir is devoted to a description, very fairly minute and accurate, of the general structural anatomy of the cerebral cortex, the descriptive anatomy of the convolutions being left to the later portion of the work. While it contains nothing particularly original, it is yet a very good *resumé* of the latest discovered facts by European investigators, and, as such, deserves a detailed notice.

After a short historical introduction, in which he sums up by giving to Malpighi the credit of first noticing the cortical cells and fibres, to Vicq d'Azyr and Baillarger that of noting the macroscopic appearances, and to Meynert, Betz and Luys the description of the microscopic structure, and a few remarks on the general plan of fissures and convolutions, the author passes to the consideration of the elemental structures of this part of the nervous centres, the various kinds of cells, pyramidal, giant, fusiform, the myelocytes, the neuroglia, the fibres and vessels, are noticed—some of them fully described and figured; and then he enters into a complete description of the five or six layers which form the cortex, following principally Meynert and Bevan Lewis in the views he adopts. Next, giving attention to the structure of particular convolutions, he notices the motor regions, characterized by the presence of giant cells (Betz, Major), and alludes to the more recent discovery of Pierret, that the dimensions of these cells are not merely in relation to their motor function, but also to the extent of the centrifugal track of the nerves of which they seem to be the origin.

The occipital or sensitive convolutions and their distinguishing differences from the motor ones are described, and an interesting parallel is drawn between their structure and that of the retina; and the author adopts Meynert's opinion that the retina, in its structure, its development, and its functions, is to be regarded as an expansion of the sensory portion of the cerebral cortex.

The special types of convolutions are next described. The first, the author here following Meynert, is that of the Sylvian fissure convolutions, characterized by a special development of the lowest or claustral layer containing the greatest number of the elongated fusiform cells. As to the special relations of the claustrum which underlies these convolutions, which Meynert considers to appertain to the cortical system, while others, such as Luys, hold that its relations are with the corpus striatum, our author expresses no positive opinion. The other special types of cerebral convolutions mentioned are the hippocampus and the olfactory bulb, the morphology of which, as well as the microscopic structure, are described in detail.

The remainder of this part of the memoir includes remarks on the minute comparative anatomy of the convolutions, on the white substance, on their circulation, and on their development. These are simply judicious *resumés* of the statements and opinions that have been advanced by various authors. M. Richet does not usually, as we have seen, commit himself on disputed points, but is suggestive and very fair in his statements. Some of the most recent papers, such as that of Pansch, on the development of the convolutions, are not noticed, though the author is generally very well up to date in his citations.

The second part of M. Richet's memoir commences with an anatomical introduction, giving the general topography of the surface of the hemispheres, and a historical introduction reviewing the physiological ideas that have been held in regard to the cerebral convolutions from the earliest period down to the present. In regard to the credit of the discoveries in cerebral physiology, it is well known there has been some controversy, but the summing up of our author seems to be eminently judicial and fair.

Passing next to the study of the physiology of the cortex, M. Richet divides it into two terms, its properties and its functions. Following out this division, he first considers the excitability of the convolutions. He reviews the controversy as to the diffusion of electric currents raised by Dupuy and others, and the relative excitability of the grey matter of the convolutions and the subjacent white fibres, and discusses the experiments of Carville and Duret, Putnam, Braun, Franck and Pitres on this point, and narrates some of his own. The conclusions he arrives at are stated as follows:

1. "That peripheral diffusion may be avoided, but that diffusion downwards through the grey matter to the white, is unavoidable;

2. "That the white substance of the brain is certainly excitable, as was thought to be the case by Haller, and denied by Flourens ;

3. "That probably the cortical grey matter is also excitable by electricity, although direct proof of this is almost impossible ;

4. "That we may admit, perhaps still hypothetically, the following order as regards excitability (electrical) of the nervous organs (*a*) nerve terminations; (*b*) nerve trunks; (*c*) grey matter of the centres ; (*d*) white substance of the centres."

Passing by what is said as to the effects of drugs on the excitability, on the thermic, electric and chemical conditions, in regard to which, M. Richet agrees with Hermann rather than Du Bois Reymond, and on the cortical circulation, all of which is a well stated and judicious summary of our knowledge of the subjects, we come to the second chapter, on the functions of the convolutions. We cannot abstract this, it is a critical summary of all or very nearly all of the investigations that have been made on the subject, arranged under the three heads of the motor, the sensory and the intellectual functions. The first of these is again subdivided as follows : First, (A) is given a brief account of the methods of investigation ; then (B) the action of the convolutions on the muscles of animal life ; (C) their action on the muscles of organic life ; (D) on aphasia, and (E) the theories of the motor innervation of the convolutions. Only the last of these needs remark, and we can best give the author's ideas in his own language, in his summary of facts.

(A) "Cortical paralyses are transitory in the dog; they are permanent in the ape (Ferrier), and in man Charcot has shown, with an imposing mass of evidence, that they exist permanently after lesion of one hemisphere, without lesion of the ganglionary centres.

(B) "Cortical (Rolandic) lesions never induce anæsthesia (immediate or consecutive) in man, while in the dog, sensibility is certainly affected.

(C) "There therefore exists a notable difference between the brain of man and that of the dog, hence conclusions drawn from the case of the one should not be applied to the other except with considerable reservations.

(D) "In man there are very rare and exceptional cases, if we eliminate the doubtful ones, but certainly some authentic cases, in which deep and extensive lesions of one or other of the Rolandic convolutions have not produced paralysis, as well as of lesions of Broca's convolution that have not caused aphasia.

(E) "Nevertheless, in a great majority of cases, a cortical lesion of the hemisphere causes sclerosis of the lateral columns, the medulla oblongata, etc., without passing through the basal ganglia. If, as is probable, the trophic action (?) and the excito-motor action follow the same route, we may conclude that there are white bundles passing directly from the Rolandic region to the cord and muscles without passing by way of the ganglionary centres.

(F) "On the other hand, it is certain that, in the dog, every excitation of the Rolandic zone not only acts (directly?) on the muscles, but also on the sensibility, the arterial pressure, the salivary secretion, the vaso-motors, etc.

(G) "In the new-born the cerebral cortex is not excitable, nevertheless they are capable of movements and of (unconscious) sensibility. After the ablation of the sigmoid gyrus in new-born pups there remains no paralysis.

(H) "Are there pyscho-motor centres? This is very dubious, but it is of little importance, and it is already a beautiful result of united physiology and pathology to have demonstrated psychomotor bundles of fibres, more developed in man than in the dog.

1. "Therefore, in the majority of individuals, but not in all, there exist well limited pyscho-motor bundles, that may be separately paralyzed, atrophied, or excited.

2. "This isolation is very imperfect in the dog; not only between the various motor bundles, but also between the cortical motor zone and the other cerebral regions.

3. "The pyscho-motor apparatus, comprising the whole cortex and the conducting fibres, is much less developed in animals than in man.

4. "It is a highly developed apparatus (*appareil de perfectionnement*) that matures slowly, and only in the higher animals."

These conclusions the author states are only provisional, and they are certainly sufficiently cautious and conservative in most respects. Nevertheless, in one point it seems to us that a statement needs modification. Tarchanoff, whose researches are alluded to in a foot-note, but not mentioned at length, has shown that there exists quite a difference in different species of animals as to the excitability of the cortical motor regions at birth, hence the general statement that the cortex is unexcitable in new-born animals is hardly correct.

The section on the sensory functions of the cerebral hemispheres is mainly only a statement of Ferrier's investigations in this direction, with some references to those of Munk, Charcot, and others. There is also nothing specially original or striking in that on the intellectual functions of the convolutions, the subject is treated very briefly, the author does not enter at any length into the speculations that are suggested by it.

In closing we will say of the work as a whole, that it is a very good critical summary of the anatomy and physiology of the cortex of the hemispheres, as far as at present known, or at least, as far as known up to a very recent period. The careful and conservative spirit of the work is everywhere conspicuous, and we are not sure that the author does not sometimes carry his conservatism a little too far in certain directions; this however, is an error on the safe side. To those who read French, and every educated physician should be able to do this, it will be found a convenient and useful work of reference.

V.—BUCKE: THE MORAL NATURE.

Man's Moral Nature. An Essay. By Richard Maurice Bucke, M. D., Medical Superintendent of the Asylum for the Insane, London, Ontario. New York: G. P. Putnam's Sons. 1879. 200 pages.

This is, in many respects, a rather curious work. Its claim for notice in a medical journal not specially given to the discussion of ethical theories is in its treatment of its subject. There is, we believe, a physiology of ethics: the moral nature of man has its connections with his physical organization, and so far as the work before us is an attempt to work out and describe those connections, and to propose and support a physiological theory of morals, it falls within the special province of this journal.

It appears to be an expansion of two papers by the same author, read before the American Association of Superintendents at their meetings in 1877 and 1878, and published in the October numbers of the *American Journal of Insanity* for the same years. These articles attracted our attention at the time, but did not appear to call for special notice. The matter has here been re-arranged, and much has been added that is entirely beyond the scope of the original articles, and, indeed, altogether outside of the limits of physiological and medical science. Nevertheless, we shall give portions of these additions such notice as they appear to demand in a consecutive review of the volume.

The first thing that strikes attention as we open the volume is the manner in which it is introduced to the reader. We do not usually read the preface of a work first; but in looking at a book with an idea of writing a notice of it we frequently give a glance at its introduction to see what the author states are its objects, its methods and its scope. In the volume before us this is divided into three parts, headed, respectively: "Advertisement," "To the Reader," and "Introduction" — a rather unusual method, to say the least, and one which savors of affectation. All three of these might well be rolled into one—or, rather, the last-named one might be left to stand alone, with better appearance than at present. The principal effect which this peculiar preface, as well as the dedication to Walt Whitman, as the man possessed of the most exalted moral nature, has had on our mind, has been to put us on our guard against any prejudice that might be excited by this indication of the author's idiosyncrasies. But we must state, after perusal of the entire work, that this peculiar manner of opening is not altogether misleading in the impression it naturally creates.

The "Introduction," however, is short and to the point. Dr. Bucke says: "The object of this essay is to discuss the moral nature—to point out, in the first place, its general relation to the other groups of functions belonging to, or rather making up, the individual man, and also to show its relations to man's environment. Secondly, to show its radical separation from these other groups of functions; then to attempt to decide of what organ it is a function—to consider whether it is a fixed quantity, or whether, like the active nature and the intellectual nature, it is in course of development; and if the moral nature is progressive, to try to find out what the essential nature of this progress is—upon what basis the progress itself rests—the direction of the progress in the past and in the future—its causes—its history—and the law of it—and to point out the conclusions which can be drawn from this progress as to the character of the universe in which we live."

It will be seen from this that this is a work of very great comprehensiveness as to scope; that it covers, in a general way, the whole range of moral philosophy. We will take up several of its chapters in detail, giving more attention to certain parts than to others, and see how far the author has accomplished the task which he has undertaken.

The first chapter is entitled "Lines of Cleavage," a metaphorical title that is somewhat objectionable, and which we should infer was adopted more on account of its sound than its sense. In it is attempted an analysis of man's nature, and here we look for at least a general definition of the subject of the work, man's moral nature. The first intimation we have of it is given on the first page, where the author says: "Man reacts upon and towards the external universe in three ways, namely, by his active nature; by his intellectual nature; by his moral nature—that is, he acts upon it, thinks about it, and feels toward it." Elsewhere in this chapter he says the moral nature belongs to the dynamic part of man's being, that it deals with qualities, and that it occupies the farthest extreme from his active nature which deals with forces—the intellectual nature, which has to do with phenomena and relations, being intermediate. Leaving aside for the present the question as to the usage of the terms here, we pass to the next chapter on "The Moral Nature and its Limits" for a more complete analysis and definition. Here we are told that the moral nature is a bundle of faculties; that its principal, if not its sole, functions are love, hate, faith and fear. These, he says, defy analysis, and they are each capable of existing alone in the mind, unassociated with any concept. Faith, he says, is the opposite of fear; it is almost identical with courage, confidence or trust, which are only names for the same thing in different intellectual connections. He goes at some length into explanations of the various emotions and passions as made up of these elements in relation with each other and with various intellectual concepts. Then, again, he says, "the intellect knows; the moral nature feels," and again, "the elements of the moral nature are moral states, most of them being what we call emotions." From these we can form, perhaps, an idea of what he understands by the moral nature: though it is said that it is made up of functions which are elsewhere said to be moral states; that it belongs to the dynamic and reactive part of our existence as contrasted with the static or receptive, and yet it consists of feelings. But apart from all the confusion and consequent misuse of terms, it is clear from the context of the passages quoted that he includes nearly or quite all of our emotional nature under this head; in fact, he leaves no room for it elsewhere in his analysis of man. This is, of course, a more general conception than is commonly admitted, and it is to be noted that Dr. Bucke leaves out of consideration here altogether what is sometimes deemed the most important element of morals in the usual and less general sense than that here employed, the feeling of duty or obligation, which he perhaps includes as a complex intellectual product; and yet it is evident that he recognizes no other kind of morals than what he includes in his term, moral nature. Our principal objections, therefore, to this chapter are its faulty and incomplete analysis, the confusion of terms, implying to some extent, also, a looseness, if not confusion, of ideas, and the great expansion and misuse of the term, moral nature, to cover the whole range of human feeling or emotion, which, as we have said before, is the natural inference from the language in many places. The chapter is well enough written, and shows some thought, but nothing new or remarkably striking in this direction.

Allowing, however, to Dr. Bucke the privilege of assigning his own limits to the moral nature, we shall see in the next chapter how he applies to it his physiological theories, and what is this physical basis of the moral nature which forms the subject of the third and longest chapter of the book. Those who have seen the articles in the *Journal of Insanity* on the moral nature and the great sympathetic, will find in this chapter a reproduction of the substance of those articles, with some additions. Dr. Bucke claims to find the physical organ of morals in the nervous system of organic life—in the sympathetic ganglia. It is in this that the novelty of his work is to be found, whatever there is of it. After a general anatomical discription of the sympathetic, in regard to which we would only remark the rather extraordinary statement as to its mass, which is said to "perhaps not fall much short of the mass of the cerebro-spinal nervous system," he begins to discuss its functions. In this the only special point of interest is his answer to the question, "Is it the nervous system of the moral nature?" He argues that it is, from the following reasons. Of course we condense his argument:

1. We feel that our emotions have their seat not in our heads but in our bodies.

2. The moral nature is more developed in woman than in man, and the sympathetic system is also more developed in proportion to the cerebro-spinal system.

3. The functions of the sympathetic are, as compared with the cerebro-spinal ones, continuous functions, while the emotions are likewise continuous, as compared with intellectual and physical operations.

4. The sympathetic is deeply situated in the body, and nowhere in direct connection with the external world; and in this respect it tallies with the emotional life, which is also deeper and more isolated from our environments than is our intellectual life.

5. The simplicity of moral states corresponds with the comparative simplicity of the sympathetic nervous system.

6. The range of intensity of moral, or emotional, states corresponds with the varying range of intensity of the sympathetic functions.

7. There is a relation between the size of the organism and the development of the moral nature. Small men are deficient morally. Now, since the size of the body depends upon the activity of nutrition, which is governed by the sympathetic, the relation of the latter is obvious.

8. The relation that exists between moral elevation and length of life. "The argument is, first, other things being equal, those who have the best and highest moral natures live the longest; second, length of life depends upon the degree of perfection of

the great sympathetic nervous system. Thirdly, therefore, the moral nature is a function of the great sympathetic."

We do not think that in condensing the thirty-seven pages that they occupy in the book into these eight simple propositions that we have done any injustice to Dr. Bucke's arguments. The propositions can stand alone to the reader, and whatever value or truth they may contain, and their fallacies, would be equally evident to the reader of the volume itself. Nor need we go into any elaborate refutation of the argument: such as it is, we have given it all the space it deserves, and will leave it to the judgment of our readers. The remainder of the chapter is a continuation of the same line of reasoning. The author takes such facts as the emotional disturbances connected with disorders of organs especially innervated by the sympathetic, such as the heart, the ovaries, the stomach, etc., to prove his position, and even forces a point to make out the emotions aroused directly through the senses, such, for example, as those excited by music, to be due to the excitation of the sympathetic system.

It is not worth while for us to follow the argument farther into details. The chapter is simply a sort of reversion to antiquated psychological ideas, prevalent in the dark ages, when physiology was unknown, and an attempt to support these exploded theories by an evidently very imperfect knowledge of the anatomy and physiology of the nervous system. The author seems entirely unacquainted with the intimate physiological connections of the sympathetic with, and its dependence upon, the cerebro-spinal centres. He speaks of it throughout as if, in its functions, it were an altogether independent factor in innervation, not at all in subjection in the higher animals to the controlling and predominating influence of the cerebro-spinal axis. We should apologize to our readers for giving so much attention to these points, but in consideration of the fact that these are the views of a very respectable physician, evidently a man of considerable general reading, a leading member of the American Association of Superintendents of Asylums, and the additional fact that they were read without contradiction, so far as we know, before that association, and received the honor of publication in its organ, they obtain a sort of importance not exactly in proportion to their intrinsic merits. But this would not justify any labored answer or correction of the errors. If any one who reads this cannot see at once how unsupported they are by the really scientific modern physiology, we would simply advise him to recommence his studies or reform his mental constitution.

The last two chapters are entirely extra-medical, and whatever their errors or merits may be, we have no call to notice them. Taking all things into consideration, we do not think the author has done wisely in the production of this work.

VI.—FOURNIER: CEREBRAL SYPHILIS.

LA SYPHILIS DU CERVEAU. Par Alfred Fournier. Leçons cliniques. Paris, G. Masson, 1879. (*Syphilis of the brain. Clinical lectures.*)

In a bulky volume of some 600 pages Fournier describes—of course in detail—the manifestations of syphilitic lesions of the brain. His style is both plain and attractive so as to make it a pleasant task to digest the book. He is essentially a clinicist himself and writes for clinicists. The pathological anatomy is only given in broad sketches and the microscopic details are not discussed.

Fournier commences the first chapter with the statement, that syphilitic lesions of the brain are very rarely referred to by older authors. It is only some thirty years that they have been generally recognized. Nevertheless they are at present of great frequency; in such affections as hemiplegia of infancy, sudden epilepsy in middle age, and paralyses of ocular muscles, syphilis is the most frequent cause.

Cerebral troubles may occur early or late, after syphilitic infection, perhaps oftenest from 3 to 18 years after the initial ulcer. Syphilis like most morbid diatheses localizes itself generally in the organ "offering least resistance;" at any rate over-use of the brain, worry or excesses are about the only tangible predisposing causes for its occurrence in the brain.

Specific changes in the brain are of two varieties: 1, the syphilitic lesions proper, and 2, the secondary consequences, such as may follow any ordinary morbid process. The first class comprises *sclerosis* and *gumma*, which may choose as seat either the meninges, the vessels or the brain substance. As characteristic of Fournier's peculiar categorical style, which, while it has its attractions, leads him now and then into rather schematic descriptions, we can refer to his definitions of these lesions: *sclerosis*, energetic hyperplasia with tendency to permanent fibrous organization; and *gumma*, degenerative hyperplasia, disorganizing from its very beginning, and ending with caseous degeneration.

The author enters thereupon into a detailed description of the essential syphilitic brain changes. We cannot but notice, however, that much of this description is rather schematic. The histological details are barely referred to. While supporting his views by extensive literary quotations, it seems remarkable that the author does not mention any of the late German researches on this topic, especially the affections of the vessels described by Heubner, Baumgarten and others.

The specific lesions set up a train of secondary consequences in their vicinity: thus a gumma or a sclerotic foyer is generally surrounded by a zone of inflammation; an obstructive lesion of a vessel is followed by white or red softening in the territory of this vessel, &c. These secondary changes have nothing specific about them and are therefore *not influenced by specific medication*, a point especially insisted upon by Fournier. He claims altogether, that patients die very rarely from the specific lesions directly, but only in consequence of the secondary change caused by the specific process. The syphilitic lesions themselves are quite amenable to treatment. Post-mortems show not infrequently traces of specific lesions, which according to the clinical history had existed in previous years, and vanished completely as far as symptoms were concerned. But if the treatment is commenced only after secondary—non-specific—changes have set in, it is of little avail The anti-syphilitic medication can remove the gumma, but *not* the inflammation to which it has given rise.

The manifestations of syphilitic changes in the brain are characterized by their diversity and multiplicity. In order to render their description more intelligible Fournier ranges the various symptoms under the following general headings: 1, the cephalalgic; 2, congestive; 3, convulsive or epileptic; 4, aphasic; 5, mental, and 6, paralytic group.

The various combinations of symptoms are sufficiently typical to justify this classification, although various stages may succeed each other or even intermingle.

1. The headache is of course the main symptom of the cephalalgic group. It is always deep-seated and is described either as steady pressure, or as intermitting pangs, like the blows of a hammer. It may be of small extent or diffuse. Although not pathognomonic, the following traits of the pain help to form a diagnosis, viz.: the extraordinary intensity, the nocturnal exacerbations and the long duration. The headache is often a premonitory symptom of more serious mischief.

2. The group of symptoms called by Fournier the congestive form is quite frequent in occurrence. The author divides it again into the transitory and the more permanent varieties.

The former is characterized by vertigo and dullness, temporary troubles in the sphere of the special senses, momentary impairment of various movements and of the intellect. Later on the symptoms become more persistent, and may then be described as the permanent form of the congestive group. If this state continues, new symptoms usually appear, which the author describes as *abortive paralyses* and *congestive attacks*. The muscular palsies receive their name "abortive" from their usual nature, being mostly circumscribed in extent, very slight in intensity, amounting only to a paresis, and of but temporary character. The congestive attacks consist either in sudden and momentary failure of muscles, without loss of consciousness, or in apoplectic fits, disappearing however often without consequences. These congestive attacks are usually of grave importance, and are sometimes followed by permanent paralyses, leading thus into the paralytic stage.

In some cases syphilis of the brain betrays itself for the first time by an apoplectiform attack. These cases seem sufficiently frequent to induce Fournier to describe them as a separate group with the heading *malignant* form. After some slight symptoms, a sudden deep coma may set in, leading either to death or recovery. In view of such instances Fournier advises us to think of syphilis in every case of sudden unaccountable coma.

3. The epileptic group of symptoms is of considerable importance, since almost every case of epilepsy *commencing after the age of* 20 years, is due to a specific cause. Spontaneous epilepsy commences rarely after youth, and other causes are also quite rare. At any rate epilepsy is a frequent symptom of syphilitic brain disease. Fournier attributes this fact to the prevalence of specific lesions in the cortex cerebri, from irritation of which parts epileptic seizures can be induced, according to the recent physiological researches.

By the clinical aspect alone syphilitic epilepsy cannot be distinguished from ordinary or true epilepsy. The appearances are identical in both. There may be either grand mal or petit mal. It may be complicated or not with other brain symptoms ; it can occur early or late after infection. Very frequently is it preceded by the premonitory headache. In such cases energetic treatment can probably prevent its occurrence. Anti-syphilitic measures, however, not persistently applied or with insufficient vigor, will only relieve the cephalalgia without preventing the epilepsy.

Nearly 100 pages are devoted to a detailed consideration of the symptoms and complications of syphilitic epilepsy. It would lengthen this review unduly were we to follow the author throughout. As an interesting point he describes some variations occurring in certain non-typical cases. They consist in complete or partial preservation of consciousness during the attack, and secondly, in circumscribed attacks affecting only a group of muscles or one-half of the body. These forms are sometimes preceded by or even intermingled with typical attacks. When the consciousness is preserved the attack is sometimes accompanied by pain, moderate or even intense, in the convulsed muscles.

As a practical remark, in which by the way the book is very rich, the author points out the occasional occurrence of epileptic seizures during the night, when they may pass off unnoticed by the patient, who complains perhaps only of lassitude in the morning.

The chapter concludes with the decided advice to institute energetic anti-syphilitic treatment in all cases of epilepsy commencing in adult life, which are not traceable to another cause. This is to be done no matter whether a specific history exists or not.

4. The syphilitic aphasia is next described in detail. Some of the more important points can be mentioned in a few words.

Although not differing in its clinical aspect from aphasia due to any other cause, syphilitic aphasia can be proven as a specific form both by the influence of anti-syphilitic treatment and by the autopsy. It occurs sometimes early, sometimes late in the history of syphilis. It may be the only symptom of brain disease, or be accompanied by other troubles, especially hemiplegia of the *right* side or paresis of the tongue. According to the lesion causing it, it is either temporary or more or less permanent.

5. Mental disturbances are a very frequent sign of brain syphilis. Fournier recognizes two types, the chronic form, so to speak, and the acute states of exaltation. The latter are not universally recognized, the former are more generally known. The slow forms of mental disturbance manifest themselves as gradual impairment of the intellect, mostly with failure of memory, combined in some cases with more or less incoherence of thought and action.

The more acute form of syphilitic insanity, the "folie syphilitique," is quite rapid in its onset, and comprises the phenomena of exaltation, delirium and mania. As a separate form of insanity, it is, however, admitted by but few alienists. In this case, also, there is nothing characteristic in the symptomatology; on the contrary, the state can simulate any acute mental disorder. The clue is only in the history; when properly treated, the disorder yields usually with astonishing rapidity. Much has been said by various authors about the relation of syphilis to general paralysis. According to Fournier, there is no etiological relationship; a syphilitic person may also suffer from general paralysis, but he is not more exposed to this disease than others by reason of his syphilis. He describes, however, a somewhat similar, but decidedly specific, affection—*general pseudo-paralysis of syphilitic origin.* This mental disorder the author compares with the previously mentioned non-specific disease, describing first their great resemblance. Hereupon, however, on analyzing the symptoms, he discovers numerous aberrations from the regular course of general paralysis. But it seems to us that these differences, striking as they may appear by a comparison of numerous cases of both disorders, will not enable the clinicist to form a diagnosis in any given case. Similarly the anatomical lesions. At first sight they appear identical; but in reality general paralysis is due to an encephalitis of the grey cortex, the syphilitic disorder to a sclerotic meningitis. But at the post-mortem it is certainly not as easy a task to distinguish between the two lesions with the unaided eye as Fournier would have us believe. The real deciding test is the microscopic appearance, which the author does not describe at all. At least Meynert, after thorough researches, claims in his oral lectures that the syphilitic lesions can be easily recognized as such with the microscope. The chief clinical differences between the two forms of insanity are, on the one hand, the history—a point not sufficiently insisted upon by Fournier; on the other hand, the curability of the syphilitic affection, and the absolute resistance of real general paralysis to all medication.

6. The paralytic form of brain syphilis is of common occurrence. In most cases the headache, congestive and other symptoms, lead finally to various paralyses. In less numerous instances the paralytic symptoms are the first manifestation of brain syphilis. The cranial nerves are attacked in the following order of frequency: The nerve most frequently implicated is the III., next in frequency are the VII., XII., VIII., II., I., and V., while the IX., X. and XI. are but very rarely involved.

The motor oculi is very frequently paralyzed by brain syphilis, the abducens nerve less often, and the trochlearis least of all ocular nerves. Perhaps, as Fournier himself admits, paralysis of the latter is often overlooked on account of the difficult diagnosis. The anatomical reason for this predilection is the long course of these nerves, exposing them to all morbid processes starting in the basal meninges or bones. In the detailed description of the ocular palsies we find nothing new; but the advice at the end of the chapter is worth repeating: "Ocular paralyses point towards syphilis, and if of syphilitic origin, indicate usually a threatening danger from some intra-cranial process."

The description of paralysis of auditory and optic nerves contains no new points, but is written in a practical way. Stress is laid, and very justly, upon the necessity of examining the fundus of the eye in all cases of brain syphilis, *even if there are no symptoms on the part of the eye.* According to Hughlings Jackson, an optic neuritis (choked disc) may exist without impairment of vision! Such a discovery in a given case can often correct both diagnosis and treatment.

Nothing further can be said of Fournier's account of paralyses of other cranial nerves and monoplegias, but that it is short, practical, as far as it goes, but entirely free from any description of finer modes of investigation, as electricity, etc.

Hemiplegia is a very frequent symptom of brain syphilis. As regards its prognosis, Fournier distinguishes two varieties, viz.: (1) hemiplegia due directly to syphilitic lesions, and (2) hemiplegia due to non-specific brain changes caused, of course, by a specific lesion in another spot. Although it is hardly possible to recognize the real condition of the brain before the therapeutic experiment, Fournier's ingenious distinction is certainly to be admired. Thus it is a serious difference to the patient whether his hemiplegia is caused by compression of a motor tract by a gumma, which can be removed, or whether the cause is a want of nutrition of a motor part by reason of obliteration of the vessels from distant syphilitic lesions.

Hemiplegia is usually preceded by syphilitic headache, and the various symptoms of the congestive group, including affection of motor and sensory nerves. Occasionally a few attacks of temporary, incomplete hemiplegia precede the regular, more or less permanent state. Syphilitic hemiplegia is usually incomplete both in intensity and extent. The sensibility is usually intact, but sometimes, though rarely, more or less impaired, and now and then perverted. Intellectual disturbances occur also in some cases. No characteristic difference can be found between an ordinary hemiplegia and one of syphilitic origin. Here also the clue is in the history. But suspicion as to the cause ought to be aroused by a combination of symptoms, especially the coexistence of paralysis of any ocular muscles. The age is also a guide, the specific form occurring most frequently before the forty-fifth year. The probability of a syphilitic origin is, however, positively confirmed or refuted by the results of treatment.

It is somewhat exceptional to find cases presenting for any length of time only those symptoms which the author ranges in a given group. The different forms succeed each other, and very frequently intermingle. Again, a class of patients exists in whom brain syphilis produces for quite a time but very vague symptoms, especially mental dullness and muscular feebleness or even prostration. Under such circumstances, a diagnosis is possible only from simultaneous specific troubles, such as exostoses, etc. But a correct diagnosis once formed, everything is gained. Another form might be called mixed. Besides many of the symptoms common in brain syphilis, there is hardly a sign of disturbance of the nervous system, which such patients cannot display at some time or other. It is the very multiplicity of unconnected symptoms which leads to a diagnosis.

The course of brain syphilis is always influenced by medication. In many instances a positive cure can be accomplished, if not, at least the occurrence of new symptoms is prevented, although the existing ones — caused, perhaps, by secondary changes — remain unchanged. Of all forms, the mental disturbances are the most resistant to medication ; so that the existence of insanity is of grave prognostic importance in any case of brain syphilis. If not treated, all syphilitic disorders have a tendency to become permanent and to spread in extent. The chapter which Fournier devotes to the ultimate progress and termination of brain syphilis, seems to us the most instructive of the entire work. It is evidently the result of careful reflection, based on an exceptionally large experience. It is, however, too full of details to be abstracted profitably.

As regards the prognosis, the chances diminish, of course, with the duration of the affection and the multiplicity of symptoms. Yet now and then a case apparently hopeless is completely restored, so that the author endorses Ricord's *bon mot:* "As regards the cure of syphilis, everything is possible, sometimes even impossibilities." An exceptional value can be attributed to Fournier's statistic of the termination of 138 cases of brain syphilis :

Death	14	cases.
Recovery with serious impairment of intellect, motion or sensation	33	"
Recovery complete	30	"
Recovery incomplete, with permanent but slight impairment of some functions	13	"
Still under observation	11	"
Unknown	37	"
	138	

Still the fearful importance of these figures is somewhat diminished by reason of a number of cases coming under his observation at a very late stage, and the fact that of the 14 fatal cases, not one had been treated properly, at the proper time. On the other hand, the statistic does not include numerous cases with slight symptoms, cured before they could develop into unequivocal brain syphilis.

There follows hereupon an instructive account of the prognosis in different forms. While the lighter forms are very amenable to treatment, all cases are characterized by a tendency to sudden exacerbations and relapses.

The last chapter (nearly 50 pages) is devoted to the treatment. The treatment should always be commenced *early and energetically, kept up with the original energy during the course, and continued for a long time after the disappearance of symptoms.*

Fournier's chief remedies are iodide of potassium and mercury. The former is given boldly by the stomach, preferably mixed with food or drink. He claims to have never met a patient absolutely intolerant to KI. Yet in such cases it might be given by the rectum. Strangely enough, he has sometimes seen that large doses were less irritant to the stomach than smaller ones. Mercury, on the other hand, is more apt to disturb the stomach, and, hence, the author favors inunctions. He commences the use of iodide of potassium in a dose of at least 3 grammes per day, raising it finally to 6–8, or even 10 grammes, daily. Larger quantities he has not found more serviceable, but only more irritant. Hg is given in the form of proto-iodide, 0.10 to 0.20, or as sublimate in doses of 0.02 to 0.05 per day. The latter dose, large as it seems, he gives without hesitation or regret, at least for a short time.

For inunctions he begins with 5.00 per day for the first week, raising this dose 8.00 or even 12.00 grammes.

The occurrence of stomatitis is to be observed, and when present it is to be treated ; but the mercury is not suspended, on account of the proper reason that the dangers of stomatitis are slight compared with the dangers which we remove with mercury.

During the continuance of the disorder the treatment is to be kept up with the original energy. But there is always some accustomation to both KI and Hg. This however lasts but a few weeks after suspension of the remedy, and hence Fournier advises strongly *alternate medication*, changing from KI to Hg and back every few weeks. This treatment is to be continued long after the disappearance of all symptoms. As auxiliary means he advises cold douches, which in his experience as well as that of Charcot, hasten the cure materially, especially in syphilitic epilepsy. All special indications are of course fulfilled by the usual palliative and other means. The cure being accomplished Fournier insists for some time on strict hygiene, *i. e.*, avoidance of all excesses, especially venereal indulgence, exertions, and brain work, his object being to give the brain absolute rest.

This incomplete review of the salient points of the book can give the reader an estimate of its value. It is the result of a large experience of a careful, logical observer It contains a good deal which cannot be found in any one work, though perhaps little entirely original with the author. It can be read profitably by everyone, especially in this country, where brain syphilis is either very rare, or overlooked. But it requires attention to read it, in order to recognize at once when the author schematizes, which he does a great deal. At any rate the title, clinical lectures, is not an appropriate one With due care one will find the book very valuable for practical purposes. But the reader will be disappointed if he expects scientific research, either in the account of autopsies, or in physiological modes of examination.
H. G.

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IV. Diseases of the Intestines and Peritoneum. By John Syer Bristowe, M. D., J. R. Wardell, M. D., J. W. Bigbie, M. D., S. O. Habershon, M. D., T. B. Curling, F. R. S., and W. H. Ransom, M. D. New York: Wm. Wood & Co., 1879.

V. Pott's Disease. Its Pathology and Mechanical Treatment, with Remarks on Rotary Lateral Curvature. By Newton M. Shaffer, M. D. New York: G. P. Putnam's Sons, 1879. Chicago: Jansen, McClurg & Co.

VI. Posological Table: Including all the Officinal and the most frequently employed Unofficinal Preparations. By Charles Rice, Chemist, Department of Public Charities and Corrections in New York. New York: Wm. Wood & Co., 1879. Chicago: W. T. Keener.

I. The report of the Commissioners of State Charities for Illinois commences with the statistical and other information in regard to the different charitable institutions, from which it appears that a careful scrutiny is given to the expenses of these establishments. A reduction of nearly two hundred and fifty thousand dollars from the amount asked for the two years which had to be provided for by the last legislature was recommended, and apparently very good reasons for the most part are given for the recommendation.

In regard to a request for one thousand dollars for apparatus for pathological investigation from the Northern Asylum at Elgin, the commissioners say, "The apparatus for a pathological laboratory would be useless without a pathologist." We regret that this is so; the medical superintendent and assistant physicians of such an institution should be themselves competent to undertake pathological investigations, and should have the time as they have the opportunity for such work. Still we doubt whether one thousand dollars would produce adequate scientific results within two years in many of the asylums of this country as they are at present organized. As regards the appointment of a pathologist for the State asylums, the commissioners say that without co-operation of all of these institutions, he cannot well be appointed, and they therefore advise the postponement of the consideration of the subject for the present.

The actual increase of insanity in the State is admitted and accounted for by the more settled condition of the population, gradually approaching the condition of older States, and being no longer made up of the more healthy frontier population. "An increase in the number of the insane is to be expected, until the ratio of deaths of insane persons to the entire population is equal to the ratio of new cases not restored to reason. None of the States in the great Mississippi valley have as yet reached that point. We have, however, seen no evidence that there is any increase in the number of new cases each year relatively to the total population."

The question of the Illinois law of commitment of the insane, which has already been noticed in the editorial columns of the Journal, is discussed at some length. The efforts to obtain a change in the law failed with the last legislature, and all the evils of the present system have another two years' lease of life. We need not repeat here our own opinions on this subject, we have expressed them elsewhere, and we hope what is stated in this report may have some good effect in the future. We would advise the State Board not to abandon the cause, but devise new plans of reform that may be more successful than the last.

Another important recommendation in the report, is that some provision be made for the criminal insane apart from the general hospitals. Our readers will probably readily recall Dr. Dewey's article on this subject in the JOURNAL last October, which is alluded to in this report.

Appendix I. contains statistical tables of the various institutions. Appendix II. is an elaborate and valuable report on the county jails of the State, in which is contained a description of every jail in the State. The facts stated, though not worse than those that could be collected in other States, are not as a whole creditable to the great State of Illinois, or rather to the local authorities who are responsible. But there is more of interest in appendix III., which contains descriptions of every county almshouse in the State. As these establishments are the receptacles of a majority of the chronic insane it is a matter of interest to our readers to learn in what manner these unfortunates are treated. In a few of the counties they seem to be well cared for and one (Cook) has a regular county asylum containing over four hundred inmates. In the great majority, however, no adequate provision is made for their care, and in some of the poorhouses their treatment is simply abominable. This portion of the report will be useful reading for the county boards throughout the State. The final appendix IV. is Dr. Wines' report on the International Prison Congress at Stockholm, which he attended as a delegate commissioned by the Governor of Illinois, concluding with Dr. Wines' paper on the Foreign Hospitals for the Insane, read before the conference of charities in this city, last month. We hope to notice this at length at a future time.

As a whole, the report is a very creditable showing for the State Board of Charities, and especially for its Secretary, Dr. F. H. Wines, whose labor it chiefly represents. We trust it will be widely circulated and read in the quarters where it will do the most good.

II. The Transactions of the American Medical Association, with the prize essay on the Surgical Anatomy of the Carotid and Innominate and Subclavian Arteries, which is included in the same covers, forms a bulky volume of over eleven hundred pages. It contains the usual collection of papers and addresses before the sections, with record of proceedings, and a large part of the volume is taken up with obituary notices of prominent deceased physicians, largely compiled by Dr. J. M. Toner, of Washington, D. C.

The only neurological papers are by Dr. Walter Kempster, of the Northern Wisconsin Asylum, one the address on Medical Jurisprudence and Psychology, and the other on general paresis. The first of these is for the most part a general compilation of recent researches in neurology, and the latter is mainly given to brief clinical histories and accounts of post-mortem appearances in the brain in some twenty cases of general paresis, observed by the author. He repeats a statement made also by others, but which we have not ourselves verified, that this disease is rare in the Western States. It may be more common on the Atlantic seaboard, but it is unfortunately by no means a rarity in Illinois, or more particularly in Chicago, where our observations have been made.

The other articles call for no special notice; the volume compares well with its predecessors.

III. These three volumes are the reprint of a standard work, which if not one of the most recent date, is still one that is valuable to all practitioners of medicine. It belongs to the very economical and useful series now being issued by William Wood & Co. at a dollar a volume. We have already expressed our opinion as to the value of this series ; the present volumes are of course uniform in style with the others and well worth the money to their purchaser.

IV. This is the latest published volume of William Wood & Co.'s dollar series. The articles it contains are mostly short and to the point, the names of their authors, Dr. Habershon, Begbie, Bristowe, Curling, etc., are sufficient recommendation to our readers.

V. This is a surgical monograph taking up a question of surgical pathology and therapeutics, and advocating a plan decidedly different from the one most in vogue. The author condemns the practice of suspension and the use of the plaster of Paris jacket, and recommends the principle of antero-posterior support as given by an apparatus which he describes at length and calls the "plaster zone apparatus." Eight cases illustrating success in the use of this apparatus are described. The work is worthy of the careful attention of every surgeon.

VI. This is the most complete dose book we have lately met with, including a very large number of non-officinal remedies. It cannot fail to be useful, and it has some peculiarities that specially add to its value, such as the brief remarks in regard to the preparations, the indications of poisonous drugs and the caution-marks as to dose, the specific gravities, etc., etc. It concludes with comparative tables of common and decimal weights and measures, actual and approximate. The only improvement we can suggest would be the naming of the principal antidotes to the poisonous drugs, which are not given. But, as it is, it is a very convenient and useful little book.

Editorial Department.

ASYLUM REFORM IN NEW YORK STATE.

AT the risk of being thought as giving undue attention to the subject of reform in the administration of Asylums for the Insane, we again notice the phases through which this movement in the State of New York has recently passed. Under the circumstances, it ought to be no matter for surprise that the effort to secure legislative investigation of the present modes of conducting asylums with a view to reform, should have been temporarily baffled, though not, as the friends of the present regime hope and intended, finally defeated.

In our last issue we printed a petition to the New York Legislature, which had been numerously signed, and which set forth the desirability of an investigation into the mode of conduct of Insane Hospitals of any or all kinds in New York State, and designating various points toward which inquiry should be directed, and which embodied finally, several more or less specific complaints as to the medical service of the hospitals in question, omitting any particular reference to their bad financial management.

This petition was presented to the State Senate on the 20th of March last. By that body it was referred to a Committee of two Senators, who after a certain degree of attention to the subjects referred to in the petition, reported adversely to the petitioners. Since then we have received, among other papers, a marked copy of the *Albany Argus*, of Monday, May the 26th, which contains a lengthy and what would seem to be an inspired editorial, devoted to a review of the report in question of the senate committee, and also of the petition to which that report relates. The committee consisted as already said of two members, one of whom, unfortunately for the petitioners, to say the least, was from the Utica district, in which is located one of the asylums against the management of which complaint had been made. The procedure of the committee seems to have been exceedingly simple. They merely invited or summoned the signers of the petition to appear, and *state any facts they had of their own personal knowledge*, or from other sources. The committee also invited, it seems, the asylum superintendents, certainly permitted them to come before them prior to the petitioners, though their names did not appear on the petition, but for what purpose, at that stage in the proceedings, it is difficult to conceive, unless to prevent the object of the petitioners from being carried out. The committee state in their report, that not one of the petitioners knew of anything of his own personal knowledge as to the internal management of any of the asylums in the State, most of them having never been in one of them. They also report that more than one-half of the signers repudiate their signatures altogether. But worse yet, the committee report, that many of the names of leading persons on the petition were fraudulently obtained, and in some cases in the worst sense of the word. The committee report (we quote from the *Argus*), that one very large mercantile firm recognize their signatures, and those of twenty-three of their employes, as on a paper pasted to the petition. This firm, it is reported, stated, that the paper bearing their signatures was originally part of a testimonial of good character which the firm and their employes had given to a departing associate. The paper with the signatures had been cut off from that testimonial and tacked on to the asylum petition. In summarizing the committee's report, the article in the *Argus* says:

"The report, after exhibiting facts like these, sums up the conclusion of the whole movement in very strong words. The committee declare the petition to be unsupported by a scintilla of evidence; they declare the action and evidence of the few who came to the front to be an outrage on the honor of a decent and respected profession; they declare the charges, so far as they can be made stand up in sight, to be not only unproved but disproved; they declare the process of getting signatures to be an abuse of the right of petition which is beyond characterization. Malice, misrepresentation and ignorance, aye, falsehood, are made the bottom motives of the whole movement, by the report. The report is very long and very elaborate. It will be one of the ablest legal and literary documents emanating from any legislative body of our time. The public interest in it is not small and, therefore, we have here indicated its scope and character. The medical interest in the report will be universal. Every medical society in the United States should have a copy. As many doctors as can get copies should get them. The medical publications should present the report to their readers."

The extreme gravity of these animadversions on the petition and its movers, becomes apparent when their names are known. The instructed medical public must judge whether gentlemen of the professional standing and character as citizens and men of science such as the real movers of the petition are, would be likely to devote themselves to the transparent trickery, fraud and falsehood, which this committee of two, probably unknown out of their own State, lay at their doors. On it appear the names of Drs. W. A. Hammond, E. C. Seguin, E. C. Spitzka, T. A. McBride, J. G. Kiernan, L. C. Gray, W. J. Morton and others, more or less widely known in the profession, not only in this country but abroad.

We would also note, that it appears, that not only the report of the senate committee, but marked copies of the *Argus* already quoted from, have been widely disseminated to periodicals and public officials.

But it was not to have been expected, that those really instrumental in framing the petition, which was so roughly handled by the senate committee, would long rest quietly under such a load as had been publicly heaped upon them. Accordingly, at a regular meeting of the New York Neurological Society, a provisional report of its committee having the petition in charge was made.

It was as follows:

PROVISIONAL REPORT OF THE COMMITTEE OF THE NEW YORK NEUROLOGICAL SOCIETY, RELATIVE TO THE SUBJECT OF INSANE ASYLUM ABUSES.

The undersigned, constituting the Committee on Insane Asylum Abuses, of the New York Neurological Society, respectfully report, that the petition prepared by them, and which contained the complaints deemed most important, was signed by many prominent physicians, lawyers and other citizens, and presented to the State Senate on the 20th of March last.

It was referred by that body to two members, Mr. Goebel, of this city, and Mr. Goodwin, of Utica, constituting the Committee on Public Health. That Committee has now made a report so unfair, one-sided, and so grossly misrepresenting the real facts of the case, that we feel called upon to solemnly protest against its being received by the profession and public as even remotely embodying the results of a *bona fide* examination. There was not even the pretence of a fair examination made, documentary evidence was excluded, the bias of the Committee was evident from the first, and they were surrounded by superintendents who had been examined the day previous, without any member of your Committee being notified thereof.

One member of your Committee who was examined at Albany, was forced, in self-protection, to protest against the passing from superintendents to the Senator examining of slips of paper containing questions desired by the former to be propounded to the witness, and superintendents were repeatedly seen to prompt that Senator. It may suffice, in possible explanation of the bias of the Senate Committee, to refer to the fact that the Senator conducting the examination is a resident of Utica, and strongly affiliated with the authorities of the asylum there located. It will be noticed that the Senate Committee is extremely careful in summing up its so-called conclusions to speak of State institutions exclusively (meaning asylums supported and controlled by the State alone), although the petition distinctly referred to all lunatic asylums within the State limits.

As further characterizing the animus of the Senate Committee, we may refer to the fact that their piece of special pleading, miscalled a report, has been extensively circulated among superintendents and their friends, although no member of your Committee has received a copy, and we have no other knowledge of this report than that furnished by the daily papers.

It is also worthy of note that the Senate Committee, although informed that much valuable evidence could be obtained in New York, where most of the petitioners resided, evidence which could not be obtained at Albany, avoided receiving this testimony, by refusing to meet anywhere but in Albany.

Your Committee has been charged by the partisans of the asylum interest with having appended names to the petition without authority, and misrepresenting its purport to signers. These charges are false in every particular; every name appended to the petition is authentic, and no one signed it without having had full opportunity to read it and become acquainted with its intent and purposes. It is true that a few of the signers became alarmed when they received a *quasi* threatening summons from the Senate Committee, and wrote that they withdrew their names, and that still a few others withdrew theirs at the personal solicitation of the superintendents, but the greater number of signers, including many prominent physicians, continue to affirm the pertinency of all the inquiries and the necessity of a full investigation.

Your Committee proposes within a reasonable period to present a detailed report, exhibiting the actual character of the testimony presented, as well as other evidence which was excluded in support of the allegations set forth in the petition. Your Committee is conscious of the fact that they require no better vindication of their position.

T. A. McBride, M. D.	E. C. Spitzka, M. D.
E. C. Harwood, M. D.	J. G. Kiernan, M. D.
E. C. Seguin, M. D.	Landon C. Gray, M. D.
Wm. A. Hammond, M. D.	W. J. Morton, M. D.

The Committee.

By this latter report from the Committee of the Neurological Society, it will be seen that there exists the gravest contradiction as to matters of fact as well as belief, between the Senate Committee and the petitioners to the legislature. And such is the attitude to-day before the public, of the movement for asylum reform in the State of New York.

The friends of the movement have been baffled, but not defeated, and taught by experience will no doubt in the future advance toward their object with better preparation, and we doubt not with ultimate success. And in this case, by the word success we mean, that an investigation will be had whatever the results may be. The asylum authorities owe it to the public, and should be all the more willing to have it made, if they are to a reasonable extent above reproach in their management, for in such a case the asylum officers would have nothing to lose and everything to gain. But in such spirit, and by such means to resist *ab initio* an investigation, as was done at Albany, only serves to rouse suspicions which no specious, partisan reports can allay or obscure.

We have something more than a fear, that asylums as a whole will not bear a close scientific and business scrutiny. A long time since, we called attention to the enormous expense of building and maintaining many of our asylums. In particularizing, we gave a review of figures from its own reports, of the financial management of the asylum at Utica, in New York State, which seemed to us then, as it does now, to require explanation. Subsequently, one of the ablest medical journals in this country—the *Medical Record*, of New York—took up the same topic, and from the reports of the asylum, drew up a financial statement highly unfavorable to that asylum, and the *Record* publicly called upon its chief officer to explain the meaning of his own figures. But to this day, no explanation has been publicly made, nor has any apparent heed been given to the matter.

People will form their own notions as to the reasons or motives for such a silence, in the face of such a dubious financial showing. And we can as outsiders, assure all whom it may concern, that the public has formed opinions as to the motives for withholding the explanations which the case so plainly requires. This is one phase of asylum reform on which the Senate Committee did not touch, and which if it could have been dealt with as speciously as was the subject matter of the petition, would no doubt have been disposed of long since. This theme however, has been considered in some of its phases in another part of this issue of the JOURNAL, in a review of the "Report on the State Charitable Institutions of New York," by the Comptroller of the State. To that article we would refer our readers for a valuable and suggestive discussion.

As we have said on former occasions when referring to the subject of this article, we have a high degree of respect for American alienists as a body, and a reasonable faith in actual asylum management, and we trust not an unreasonable skepticism as to the reliability of the same. But we feel certain there is need, in many cases, for reform, and that the only way to be able to say just where and to what extent, is by a searching investigation. And the only defensible motive for preventing, or smothering investigation, apparent to us, would be found in the personal and unjust spirit which might dictate and pervade it. Or, if the asylums were private property, as they are not, there would be personal reasons for resisting examination. But there is no sufficient ground for such motives in the present case. We earnestly hope that the asylum officials in New York may themselves take the initiative in opening the doors of their hospitals to a thorough examination as the only way to a permanent peace in the discharge of a high and delicate public trust.

THE Fifth Annual Session of the American Neurological Association was held in New York, June 18th, 19th and 20th. Papers were read by Drs. Hammond, Beard, Seguin, Edes, Spitzka, Schmidt, Putnam and others, and the proceedings and discussions were throughout of interest. The following officers were elected for the ensuing year:

President—Dr. F. T. Miles, of Baltimore.
Vice-President—Dr. R. T. Edes, of Boston.

Secretary and Treasurer—Dr. E. C. Seguin, of New York.

Council—Drs. F. K. Kinnicutt, of New York, and Dr. L. C. Gray, of Brooklyn.

This Association may now be said to have reached a period of healthy infancy, and the prospects of its growth and continued and increasing usefulness were never better. It was our intention to have given in this number a full official report of the proceedings of this last meeting, but, owing to the failure to receive the notes in time, it does not appear. We may publish it in full in our next number.

This number of the Journal would have been received by its subscribers some two weeks earlier but for the delay in waiting for the Proceedings of the American Neurological Association, which failed after all to reach us in time for insertion.

Periscope.

a.—ANATOMY AND PHYSIOLOGY OF THE NERVOUS SYSTEM.

TACTILE HAIRS.—The following is an abstract of a paper on "Tactile Hairs," by Dr. Lester Curtis, before the Illinois State Microscopical Society, and for the report of which we are indebted to the courtesy of the Secretary, Mr. Ernest Stuart.

"The tactile hairs, commonly called whiskers, are peculiar to carnivorous animals. The growth of an ordinary hair takes place by the multiplication of the cells which surround the papilla, and the follicle possesses no muscles of volition or special arrangement of nerve fibres.

"The first noticeable peculiarity of the tactile hair, as in the snout of a mouse, is the great size of the follicle—several times that of an ordinary hair follicle; this follicle has a bulging, about one-third of the distance from the top, which corresponds with a remarkable enlargement within.

"Two-thirds of the lower portion of the follicle is surrounded by striated muscular fibres, which appear to arise from the side of the follicle by a short tendon encircling it, and these pass off to a considerable distance, to become lost in the muscular fibres of the lip. These muscles are furnished with a rich plexus of unusually large capillaries. Other muscles are attached to the hair in a manner somewhat similar to the ordinary erectors of the hair, but are more abundant and of the striated variety.

"Within the follicle and outside the root-sheath of the hair, is a close plexus of very fine capillaries, which send branches over the enlargement mentioned above, and form a peculiar ring-like plexus above it. Between the branches of this ring-like plexus, there is but little tissue; but in the mouse and cat, at least, it does not form a sinus, as is claimed by some.

"The enlargement surrounds the hair excentrically, and consists of two parts, the outer of which, when the follicle is divided longitudinally through the centre of the hair, is nearly circular in outline, and is attached to the outer part by a short, thick pedicle. It is interspersed with oblong nuclei. The inner part is not so wide, but is longer, and surrounds the root-sheath like a band."

The speaker considered these last organs to be of a nature similar to the terminal organs of nerves found in other portions of the body; but was not prepared at present to prove his position.

The paper closed with a brief bibliography of the subject, after which numerous slides were shown, in illustration of the organs described.

THE INNERVATION OF THE UTERUS.—In a long paper in *Virchow's Archiv* (Bd. 76, Heft 1., p. 1), Prof. A. Röhrig describes his researches on this subject, being partly a confirmation of previous statements, partly new additions.

As the best animal for the purpose, the author used virgin rabbits, the uterus of which responds readily to stimuli, but does not show spontaneous movements. The animals were poisoned with a dose of curare, just sufficient to necessitate artificial respiration.

On irritating various parts of the nervous system, by insertion of needles connected with an induction coil, it was found that movements of the uterus could be obtained by stimulation of almost any part of the brain or cord. The movements were, however, more energetic from stimulation of the lower parts of the cord than from the upper portion or brain. Irritation of the lumbar cord produced the most intense tetanic uterine contractions.

All motor nerves of the uterus originate in the cord below the *tenth* dorsal vertebra. Irritation of the spinal segment above this height is of no influence; while below this point the cord, if irritated, induces violent contractions. Irritation of nerve centres is very conveniently effected by supplying them with venous blood. On ceasing the artificial respiration in the curarized animal, the cord of which was severed at the level of the tenth dorsal vertebra, violent uterine contractions of the uterus were seen. These contractions continued often for the space of one hour after the death of the asphyxiated animal. This result can explain the occasional occurrence of a post-mortem parturition in pregnant human females. It may be supposed, however, that the venous blood can cause these movements by irritation of the uterus itself. In order to decide this matter, Röhrig destroyed the lumbar cord, by inserting a hot wire into the spinal canal. After this destruction no further uterine contractions could be induced by asphyxia, although the uterus still responded to direct electric stimulation.

Similar uterine movements follow the sudden checking of the blood supply by compression of the aorta. These also depend on an irritation, and hence the integrity of the lumbar portion of the spinal cord.

Uterine movements can also be brought about by injection of certain poisons into the veins. These are the various spinal stimulants, which either stimulate directly, or, at least, exaggerate immensely the reflex excitability of the cord. Their influence on the uterus ceases, of course, on destruction of the lumbar cord. Strychnia and picrotoxin are the most powerful agents; nicotine and carbolic acid act only in larger doses. The latter requires a quantity of 0.20 grm. injected into the jugular vein. Caffein acts similarly; but its effects cannot be demonstrated on a virgin uterus; it requires an organ more irritable by previous impregnation.

The promptest action can be attributed to ammonia. An injection into the vein of 5 to 8 ccm. of a two-per-cent. solution, causes an immediate powerful tetanic contraction, which lasts, with short intervals of rest, for an hour or more, changing hereupon to peristaltic movements. If the animal is killed by an overdose of ammonia, the contractions can continue for an hour after death. But ammonia differs from the other agents in stimulating the uterus itself, or, at least, its intrinsic nerves, since the contractions caused by ammonia are *not* avoided by destruction of the cord. It had been known previously that the agent is a powerful stimulant to striated muscles, but this result shows that it calls into activity unstriated muscles as well. The action is produced also by the salts of ammonium, the chloride and carbonate.

Stimulation of the uterus centre is also produced by calabar bean and ergotin. The latter, however (extr. secal. cornut. aquos. Pharm. Germ.), does not act when injected under the skin, since it is not sufficiently absorbed. The movements thus produced are not as violent as those from the previous poisons; they are not tetanic in character, but peristaltic, and persist much longer than the more violent tetanus of strychnia.

The study of the influence of chloroform on the reflex activity of the uterus, was hindered by the difficulty of chloroforming rabbits without killing them. It could, however, be observed that the influence on the uterus is about the same as that of hydrate of chloral, an agent much safer of application.

The narcosis retards immensely the occurrence of reflex contractions of the uterus. They occur not only later, but are also feebler, while the uterine centre is readily exhausted. Morphine and opium do not possess this paralyzing influence. Atropin, on the other hand, reduces considerably the excitability of both uterus and uterus centre.

The supposed action of various purgatives, and so-called *abortifacients*, has never been properly studied. An intravenous injection of aqueous extract of aloes or colocynth causes, within ten to fifteen minutes, peristaltic movements of the bowels, often finishing with defecation. Some three to five minutes later feeble, creeping constrictions are noticeable at the cornua of the uterus; but they do not develop into proper peristaltic contractions. A more powerful action is, however, exerted by extr. sabinæ. The injection of 1 grm. of extract of savin caused, within two to three minutes, strong tetanic contractions, which changed gradually into peristaltic movements, continuing sometimes for two hours. This agent acts through stimulation of the cord; destruction of the latter removes its influence. The effect, however, on the uterus was such, that Röhrig recommends highly its experimental trial in obstetrics.

The uterus centre is finally very susceptible to reflex excitation, uterine tetanus being the immediate result of any irritation of sensory nerves. This can even be produced by irritation of the skin by cold or heat.

Searching the course of the motor nerves of the uterus, Röhrig examined, in the first place, the pneumogastric and dorsal sympathetic nerves. Both possessed no influence whatsoever on the movements of the uterus. The positive results, on the other hand, can be stated in the author's own words:

1. Section of either the nervi uterini (sympathetic) or the sacral branches, causes a momentary peristaltic contraction.

2. Electric irritation of the cord (or asphyxia) causes, after section of the uterine nerves, a feeble peristaltic contraction. The same is produced by reflex excitations.

3. The same excitations act, however, more powerfully if the sacral branches have been divided, instead of the uterine nerves.

4. Section of both sets of nerves cuts off the uterus entirely from the influence of the cord.

5. Feeble electric irritation of the peripheral ends of the divided uterine nerves, causes tetanus of the organ;

6. While a weaker contraction is induced by stimulating the sacral branches.

7. Sensory fibres are found in both sets of nerves.

Examining the nerves of the ovary, Röhrig found them to contain only centripetal fibres. Irritation of the peripheral end of the divided nerves produced no visible symptom; but irritation of the central end, or of the ovary itself, does not cause any pain whatever. The centripetal nature of these fibres reveals itself only by a rise in blood-pressure. H. G.

FUNCTIONS OF THE BRAIN.—A. Musehold, at the instigation of Prof. Leyden, publishes a memoir (Berlin, 1878, Abstr. in *Cbl. f. med. Wissensch.*, No. 14) on the result of partial extirpation of the cerebrum in pigeons. The operation was performed in part after the method of Goltz by washing out parts of the brain substance by a stream of water, and in part of the cases by simple removal of the cerebral tissue with a sharpened ear spoon. Simpler results followed the experiments performed by the last named method, especially was the generally observed weakness of the extremity of the opposite side wanting in the first series. If, in the second series, the loss of brain substance was in the anterior portion of the hemisphere, no results of the extirpation could be demonstrated, while disturbances of sight on the opposite side, regularly followed the abstraction of larger or smaller portions of the posterior half. The disturbance of vision was evidenced by the fearlessness of the animal, when objects that before frightened it were brought near the affected side, in its inability after the darkening of the eye that still possessed its power intact, to pick up or recognize its food, and in its disinclination to fly, or to stop when once aroused to flight. The visual disorder was the less in proportion as the quantity extirpated was small, but in all cases, recovery occurred sooner or later, though to be sure it was never the case that the whole posterior hemisphere was removed. If, after recovery, a considerable portion of the posterior hemisphere of the other side was also removed, there did not follow disorder of vision on both sides, as would be expected if the restoration of sight was due to vicarious action of one hemisphere for both, but there was again unilateral (crossed) visual disturbance. Hence it was concluded that, after partial extirpation, the parts surrounding the portion of brain substance removed from the posterior hemisphere could learn the functions of the portion lost.

THE DEVELOPMENT OF THE BRAIN AFTER BIRTH.—At the session of the Societé de Biologie, Paris, Mar. 30 (rep. in *Gaz. des Hopitaux*), M. Parrot made a communication on this subject. It results from his researches, that the brain of the new-born is a very imperfect organ developing itself in a very slow manner. Without wishing to follow the development in all its phases,

M. Parrot spoke only of that in the first year of life after birth. He would not give attention to the general morphology nor to the chemical or histological changes occurring during this first year, but would only notice what could be observed with the naked eye.

He had made ninety-six autopsies of infants at periods between birth and the close of the first year, but principally on those between two days and four months of age. Those infants of whom he had not the real age were left aside. He divided his study into several parts, a first series including infants from one to fifteen days old, and the others passing from month to month. The following is his method of procedure: he places the cerebral hemisphere on a table on its internal face, and makes sections parallel to that face, beginning only four or five millimetres from it, and the others two to three millimetres apart.

This study is grounded on two orders of considerations, the topographical and the chromological. The following are the general results. In a topographical point of view, when any portion of the nucleus presented any quality whatever, that part which is above it exhibits inferior qualities. In a chromological point of view, there exists a constant relation between the colors of the various parts at the same age.

Taking for a centre the system of Rolando, in which appears the white substance, we may say that this system divides the brain into two absolutely different regions, anterior and posterior, the former developing more slowly than the latter. We may conclude from this, that the development of the white substance is correlative with the grey. M. Parrot observed, besides, that the precocity of development of the occipital region is in relation to the precocity of the bony sutures of that region. After having investigated the relations that exist in the development of the two hemispheres, M. Parrot says that in four-fifths of the cases it is more pronounced in the right than in the left. He observed also, that the cerebellum is much more rapidly developed than the cerebrum. In the brain, therefore, it is the cerebrum that is last developed and its anterior portion more slowly than its posterior. These results seem to be in accordance with the views now generally accepted as to the functional importance of the frontal lobes.

In reply to an objection of M. Luys that he had not taken enough into account the respective weights of the two hemispheres, the left being the heaviest, M. Parrot said that he used this test much, but could not infer that the heavier of the two hemispheres, if there was any difference, was the most developed, but rather the contrary.

THE TONUS OF STRIATED MUSCLES.—Dr. S. Tschirjew (*Archiv f. Anat. und Physiologie*, 1879, Hft. I. & II., p. 78) precedes his experiments on this subject with a resumé of his previous researches on the tendon-reflex. He had found that the muscles are connected with the cord not only by motor but also by centripetal fibres, following the usual course of sensory filaments. These centripetal fibres are the afferent route of the tendon-reflex. Histologically, Tschirjew has observed a rich net-work of nerves in the muscular aponeuroses. Since no sensory fibres can be found in the muscles, the author concludes that the nerves ending in the aponeuroses are the only centripetal fibres of muscles and not specifically sensory nerves.

Since the tendon-reflex is caused by the sudden stretching of these nerves, the question arises, whether their continuous tension can maintain the muscular tonus. In order to decide this point, a rabbit was narcotized with morphia and its crural nerve isolated. Great care is needed to avoid injuring the nerve, since the aponeurotic nerves are very easily rendered inactive. The experiment was only continued if the tendon-reflex could still be produced after the isolation of the crural nerve. The leg was now firmly attached to the board and the patellar tendon severed from the tibia and connected with the lever of a myographion to which different weights could be attached.

After this mode of preparation, the myographic record showed distinctly that division of the crural nerve is followed by a lengthening of the muscle, in other words, that the stretched muscle is in a state of tonic contraction as long as its nerve is uninjured. The extent of lengthening after nerve section depends on the weight stretching the muscle. Beyond a certain state of tension the muscle will no longer lengthen.

As an incidental but significant observation Tschirjew mentions in this place the difference in the myographic curve of this muscle preparation, when irritated by a single induction shock before and after division of the nerve. The muscle severed from the cord, contracts and then relaxes suddenly, performing several elastic oscillations after its relaxation. If, however, the muscle is still connected through its nerve with the spinal cord, the relaxation after a momentary contraction is gradual, and elastic oscillations occur. The muscle is evidently in a tonic state during its gradual relaxation.

The existence of a constant tonus from central innervation can be no longer defended. It can readily be observed, that the muscles when not stretched are completely relaxed. Tschirjew's results show, however, that when the muscles are stretched they undergo a tonic contraction lasting as long as the stretching.

This arrangement is undoubtedly of considerable mechanical importance, since thereby a condition comparable to the dead stop of machinery is avoided, as well as the elastic oscillations which muscles show after severance from the nerve centre. The possibility of a more delicate graduation of voluntary muscular efforts is thus guaranteed.

THE COURSE OF PERIPHERAL NERVE FIBRES.—In embryos of rabbits, 3 centimetres in length, Dr. L. Löwe (*Centralblatt*, No. 19, 1879) claims to have demonstrated a visible difference between sensory and motor nerves. The embryos were stained by immersion in ordinary carmine, whereupon the motor nerves assume a *darker* coloration than the sensory fibres. This deeper hue of motor nerves depends on a more thorough imbibition of their substance with carmine and on the presence of round cells in their substance. As yet, Löwe has not been able to determine, whether this difference is due only to a more advanced state of development of one or the other set of nerves, or whether it indicates a structural difference.

On account of the different coloration it can be readily seen, that in the case of spinal nerves the sensory root is lighter in color in front of the spinal ganglion, than after it has passed through the same. In the mixed trunk the sensory bundles are always found on the external side of the motor fibres, except in the posterior branches of the spinal nerves, where this relationship is reversed. According to its coloration the nervous twig passing to the nearest sympathetic ganglion seems to consist purely of sensory fibres.

THE SINEW-REFLEX.—Prof. Berger, of Breslau, publishes in the *Centralblatt f. Nervenheilkunde*, No. 4, Feb. 15, the results of his investigations on the tendon-reflex. While his observations in the main agreed with the statements of Westphal and Erb that this reflex is lacking in ataxia, he yet found two well marked cases that were exceptions to this rule, out of eighty-two examined, or 2.4 per cent. In this number, moreover, he excludes all cases not absolutely uncomplicated, especially rejecting those in which the lateral columns were involved. Moreover, there were in the eighty-two, four cases in which the patellar reflex was lacking only on one side (3 times left, 1 right) without any mentionable difference in the way the two limbs were involved in the other symptoms. In one of these cases the foot phenomenon was markedly present on both sides, and in another, only a few weeks before, the presence of the patellar reflex on both sides was demonstrated.

In order to test the question of the presence or absence in healthy individuals, he, with the assistance of Drs. Köbner and Kroner, examined over fourteen hundred healthy persons, among them nine hundred soldiers. The results are stated as follows:

1. The intensity of the patellar sinew-reflex varies within very wide limits in healthy persons. In very many cases it is so slightly developed that it is only with special care that it can be called out. Thus, numerous cases were brought in which it was said to be lacking, but in whom it was finally observed. On the other hand, in very many cases it is remarkably developed, so that only a slight tap with the finger will call forth a powerful contraction, and not infrequently a series of rapidly succeeding flexion and extension movements. Here, we were able to demonstrate, by Erb and Westphal's method, the clonic form of the patellar reflex. It does not appear positively allowable, from the facts, to speak of a pathological increase or diminution of the patellar sinew-reflex. Only the absolute absence of this (with certain exceptions) and the evidence of an exalted sinew-reflex in various other muscles in which, under normal circumstances, the contractions are neither regular nor very marked, justifies the conclusion of an abnormal condition.

2. The patellar sinew-reflex was lacking in twenty-two healthy individuals, lor in 1.56 per cent. On account of the clinical significance, I will not negect to especially state that we examined these twenty-two individuals—not

merely once, but many times—and with all possible precautions. At no time was even a trace of the phenomenon detected. Of course they were further examined in all possible directions: they appeared perfectly healthy (thirteen of them being active soldiers); there was no local anomaly in the knees; the electric contractility of their quadriceps (this only tested in a part) was perfectly normal. This result was so unexpected by me, since I had for years examined healthy persons, and always found this reflex present, and it was so constant during repeated examinations that it seems, contrary to the hitherto accepted notion, that it may be wanting in perfectly sound persons. A unilateral absence of this reflex in healthy persons has not yet been met with by me.

3. Of all the other sinew-reflexes, that from the tendo Achillis is by far the one most easily demonstrated; nevertheless, we found it lacking in twenty per cent. of the cases, and frequently existing unilaterally. On the other hand, the "*foot phenomenon*" (the characteristic reflex clonus of the foot) was only very exceptionally present in healthy individuals. I have met with this in only three young men, but in a very marked manner, and one of these had formerly suffered from convulsive tic. All three were not at all astonished at the very pronounced phenomenon; it had been familiar to them from childhood, that if they sat with the heel raised and only the toe of the foot touching the floor, there would be a tremor of the limb. The adductor reflex, and that from the tendons of the triceps and biceps brachii, have been investigated by us in only a limited number of cases (445 times in 364 persons), and we found the former present in only five per cent. (two times only unilateral, and once bilateral contraction from unilateral excitation), while the latter could not be demonstrated in thirty-five of the persons examined, or in twenty-five per cent. Besides these, I have examined many other tendons in healthy individuals without yet having ascertained definitely the proportion of their occurrence. Therefore the patellar sinew-reflex, and perhaps also the Achilles tendon-reflex, are to be principally looked for in testing the tendon-reflexes.

4. Of the various detailed results, I will only mention that we saw several times a contraction of the quadriceps follow mechanical irritation of the adductors, and, once, contraction of the adductors from percussion of the patellar tendon of the opposite side. I had previously found and described a similar condition in hemiplegia.

I have proven the exalting influence of strychnia (subcutaneously) on the quickness of the sinew-reflex, and also the quieting effect of morphine on the same. Westphal, in his latest memoir, has held that the testing of the patellar reflex is of special importance in those cases in which tabes begins with spinal amaurosis, the so-called white atrophy of the nervus opticus. Under certain circumstances the answer to the important question, whether this is an independent disorder or only a prodrome of a developing posterior spinal sclerosis, ought to be made possible by the examination of the patellar reflex.

In my hospital department of the city poor-house there is a woman, twenty-seven years of age, now blind for seven years from optic atrophy, with completely absent patellar and Achilles reflexes. The most careful examination does not reveal a single subjective or objective symptom of spinal disease: the patient moves briskly about, and is active in housework. Since it is well known that tabetic amaurosis may precede all other symptoms for years—Charcot, especially, speaks of cases in which the ataxic symptoms revealed themselves ten years or more after the atrophy— I cannot, therefore, yet assume in the above case, that there is a posterior sclerosis from the absence of the tendon-reflex. This view would have some basis, since this disease may be absolutely without symptoms up to the loss of the tendon-reflex, provided we could in this case consider this absence as pathological. I took occasion, last summer, with the friendly co-operation of the attending physician, Dr. Reichelt, to examine the inmates of the Blind Institute. There were at the time eighty-four inmates—nine from atrophy of the optic nerve. Of these, otherwise healthy, nine individuals, between fourteen and eighteen years, there were *two* in whom the patellar reflex was wanting, while out of the seventy-five others, blind from various eye disorders, it was lacking in only a single case. In this connection, I will say that in three powerful adults suffering from optic nerve atrophy, and affording no symptoms of spinal disorder, the patellar reflex was very marked in two; and in the third, on the contrary, it could not be demonstrated. This rather remarkable proportion may, however, very easily be accidental, and only much larger series of cases will allow a correct judgment. In four cases of diphtheritic ataxia (after previous paralysis of velum palati), with perfect integrity of the cutaneous and muscular sensibility, I found an absolute absence of the patellar reflex, while the cutaneous reflexes were notably present. In a shorter or longer time after recovery, in one case in about four months, the sinew-reflex was generally restored. The disturbances of co-ordination, therefore, of the lower extremities afford no resemblance to those of tabetic ataxia—apart from the Romberg's symptom, observed in all four cases—but are, rather, comparable in many respects with cerebellar ataxia (drunken gait).

With our deficient acquaintance with the anatomical basis of ataxias after acute diseases, I can only say that also in cases of co-ordinatory locomotor disorders, without the characteristics of tabes dorsalis, the patellar tendon-reflex may be wanting. The rapid recovery shows, nevertheless, that we do not here have to do with a degenerative alteration, and therefore the pathological failure of the tendon-reflex by itself does not necessarily indicate a serious tissue alteration of the lumbar cord.

Observations of diphtheritic ataxia have led me to question to what extent, also, lesions of the central nervous system situated outside of the lumbar cord may suppress this tendon-reflex, which lesions, according to the experimental researches of Schultz and Fürbringer and, more recently, Tschirjew, must be in the lumbar cord, and, according to Burkhardt, on the other hand, in the plexus or spinal ganglia. But this I reserve for a future communication. In conclusion I relate an anomalous condition of the patellar tendon-reflex, in a case which I consider as one of Remak's *tabes basalis* (optic muscle paralysis — atrophy of optic nerve producing gradually total blindness, anæsthesia of trigeminus; after some years characteristic spinal symptoms, still no anæsthesia). Here there regularly occurred, from tapping the lig. patellæ, a considerable flexion movement of the hip, knee and foot; sometimes, also, a contraction of the adductors of the thigh, but never of the quadriceps; and the involuntary movement, which could not be suppressed by the patient, was remarkably slow in making its appearance, even two or three seconds. Eulenburg has reported a somewhat similar case.

THE COMMUNICATIONS OF THE VENTRICLES WITH THE SUBARACHNOID SPACES IN THE BRAIN.—The following are the conclusions of a paper by Prof. Marc Seé, *Revue Mensuelle*, April:

1. The cerebral ventricles are in direct connection with the subarachnoid spaces of the encephalon and cord.
2. This communication takes place at all points where the pia mater penetrates into the interior of the encephalon.
3. The foramen of Magendie has not the importance attributed to it by him, and its obliteration, normal in certain species of animals, causes no disorder in the mechanism of the circulation of the cephalo-rachidian fluid.
4. This circulation should not be conceived as that of a fluid mass flowing in full tide from one cavity to another through perfectly free orifices, but rather as a true filtration through the meshes of connective tissues that support the vessels of the choroid plexus and the superior and inferior choroidean curtains.

Among others the following articles have lately been published on the Anatomy and Physiology of the Nervous System:

GOWERS, Note on a Reflex Mechanism in the Fixation of the Eyeballs, *Brain*, April.—LUDWIG and LUCHSINGER, Innervation of the Heart, *Centralbl. f. d. med. Wissensch.*, No. 23.—LEWASCHOW, On the Influence of the Crural Nerve on the Lumen of the Vessels, *St. Petersb. med. Wochenschr.*, No. 16, May 3.—SCHADOW, The Sensibility to Light of the Peripheral Portions of the Retina in Relation to Space and Color Senses, *Pflueger's Archiv*, XIX., x. and xi.—LAUTENBACH, The Physiological Action of Heat, *Jour. of Physiology*, II.—OTT, Physiology of Spinal Cord, *Ibid.*

b.—PATHOLOGY OF THE NERVOUS SYSTEM AND MIND; AND PATHOLOGICAL ANATOMY.

Ocular Symptoms in Diseases of the Central Nervous System.—The following are the principal conclusions of a recent memoir by Coingt (Paris, 1878) as abstracted in the *Centralblatt f. d. med. Wissensch.:*

Ptosis occurring by itself may depend upon an intra-cranial lesion, perhaps upon a direct implication of the third nerve or its nucleus. It commonly only affects the eye of the side opposite the hemisphere involved, where it occurs indicating cerebral involvement; and in an individual complaining of no other symptoms, the suggestion is of cerebral syphilis, or grey degeneration of the posterior columns of the cord, or chronic softening of the brain.

As regards the conjugate deviation of the eye and head, the author holds to the view of definite cerebral centres for these movements. Every excitation of one of these bilateral centres causes a conjugate movement toward the opposite side; every implication of their function has a conjugate movement toward the diseased side as a consequence. Both centres (for eye and head) are certainly very closely connected in the hemispheres, but can, nevertheless, be put into action independently. The symptom in question is observed in apoplexy, epilepsy and hysteria, and may be produced by slight as well as by serious brain lesions. It indicates (epileptic conditions excepted) a more or less serious cerebral alteration, which may be either multilocular or diffuse, unilateral or bilateral; but one side generally predominates. If the lesion is in the isthmus cerebri the deviation always occurs toward the side opposite the lesion. In lesions of the hemispheres the direction of the deviation varies: if there is hemiplegia, with or without contracture, but without special convulsions, the patient (according to Prevost) looks to the side of the lesion; are convulsions present, he looks away from the same side. But cases occur in which neither of these rules will apply.

As regards the condition of the pupil in lesions of the nerve centres, the author first notices the frequently observed inequality in general paralysis, the fact mentioned by Vincent, of its imperturbability to light. In unilateral injuries of the cervical cord the pupil is sometimes contracted, or the reverse, according to the predominance of irritative or paralytic conditions. Contraction of the pupil with coma is met with in ventricular hemorrhages, in hemorrhages of the pons, and especially in lesions of the middle brain; the pupil is sometimes narrower, sometimes wider, on the side of the lesion than on the other; most frequently, according to the author, the dilatation is on the side corresponding to the lesion.

In tabetic patients, contraction of the pupil with indifference to light is commonly observed, a combination that, according to Vincent, is not at all, or very rarely, observed in other forms of chronic myelitis.

Paralysis of the orbicularis oculi muscle is not commonly supposed to occur from cerebral cause in hemiplegia, but, according to the author, if one seeks to raise the lids of both eyes, the resistance is by far less on the paralyzed side than on the other; paralysis is therefore present, but is generally imperfect.

In conclusion, the author mentions the alternate hemiplegias, in which the limbs of one side and the oculo-motor and abducens of the other are paralyzed, which form of paralysis is well known, is characteristic of lesions of the pons.

In regard to conjugate paralysis of the abducens of the one, and of the internal rectus of the other side, the author only repeats the old Féréol-Foville observations.

PARAPLEGIA IN SYPHILITIC SUBJECTS.—Dr. E. C. Seguin, *Archives of Dermatology*, April, furnishes an interesting paper on the subject of so-called syphilitic paraplegia, a title to which he objects, and gives the details of six cases. We extract his concluding remarks:

"These six cases (and I might have added others) suggest several lines of thought, which I shall briefly follow:

"Can we, at the present time, make a positive diagnosis of 'syphilitic paraplegia?' I think not; the diagnostic argument is fragile, and includes the fallacies of coincidence, interrupted sequence, and of unknown factors. Prominent among the reasons given for such a diagnosis is the one that the patient has had syphilis. This, in view of the complexity of the morbid conditions which cause paraplegia, and of the fact that symptoms are usually (in the nervous system) caused not by the nature but by the location of a lesion, is well-nigh worthless. Of more importance is a reasoning by exclusion, demonstrating that the paraplegia under consideration differs in notable particulars from that caused by well-known lesions of the spinal apparatus. I think that it may be learned from the cases related, and from those recorded in books and periodicals, that paraplegia of syphilitic origin is often atypical. In some of my patients pain in the back or in the side was severe, and suggested a lesion of the bones, or meninges. The co-existence of a cerebral lesion, as shown by palsy of the third or sixth nerve, or dementia, or recent epilepsy, would go far to establish the diagnosis. The age of the patient, adult life, is an aid.

"It is proposed to settle this obscure diagnosis by reference, *post hoc*, to the results of treatment. One objection to this is the logical objection that it throws the diagnosis out of the proper time; it carries it forward to a time when the patient will not want it much: *i. e.*, when cured or permanently paralyzed. Another objection is that it is far from proven that no other than syphilitic affections are cured or benefited by mercury and iodide of potassium.

"This last objection involves a question of so great medical and social importance, that I feel obliged to trespass a little longer on your patience to say a few words about it. It has happened to all of us, I dare say, to witness the cure of serious cerebral symptoms by the iodide of potassium in persons who denied having had syphilis, and who bore none of the marks of the disease. I have seen at least five such cases, and in several of these there could be no question of the patient's truthfulness. The conclusions drawn from such a reasoning is usually that the affection treated was syphilitic; that the patient has deceived either himself or us as to infection.

"In the same way we are apt to argue about obscure symptoms which disappear under the use of quinine; they must be, they are malarial. There was a time when I accepted this argument as valid, but in the last two or three years a doubt has grown up in my mind, and acquired strength by added experience. This doubt is partly the result of clinical experience, and partly from an invincible objection to the doctrine of specifics.

"I have seen cases of disease which I cannot say were syphilitic, get well under iodide; and, on the other hand, I cannot be persuaded that this medicine or mercury acts specifically upon the syphilitic poison, be it in the shape of a dyscrasia or localized in solid tissues. These medicines, and others, cure disease, it seems to me, by changing and increasing the nutrition of the tissues, or by modifying the action of the nervous system, or by changing the chemical properties of the blood.

"The treatment of paraplegia which is supposed to be caused by syphilis, should be very energetic. The subject should, as soon as possible, be placed under the influence of mercury, by what method matters little. If the digestive organs are in good order I give the medicine by the mouth, guarded by opium, if necessary. The proto-iodide, the iodide, the two chlorides, and blue mass seem to be sufficiently good in proper doses. When there is no doubt as to the strength of the digestive organs, it is better to use inunctions of ungt. hydrargyri or of the oleates.

"This treatment alone may do good, but it is to be aided by the simultaneous use of the iodide of potassium, given according to the American method, viz., fearlessly in doses gradually raised from 2 grams to 24, or even 32 grams a day. When largely diluted, this seldom disturbs the stomach; much less often than most practitioners think.

"Important points in treatment of severe cases, are:

"1. To keep the bladder empty, and to prevent or reduce cystitis. This is to be done by removing the urine two or three times a day by means of perfectly smooth soft catheters, which are to be kept in carbolized water when not used. It is probable that some cases of cystitis arise by the introduction of bacteria into the bladder by dirty catheters. If cystitis exist, injections of lukewarm water, of borated or carbolized water, will do good, or even cure the disease.

"2. To prevent bed-sores, by keeping the sheets and shirt of the patient perfectly smooth and taut; by preventing urine from running under him; by frequent sponging with alcohol and water, and by the use of powders. If bed-sores have formed, they should be treated by ice or snow poultices for ten minutes twice a day, and stimulating dressings during the rest of the time; all gangrened shreds should be picked out, and the recesses of the sore injected with strongly carbolized water. Pressure should be removed by change of posture and by appropriate pads.

"In the stage of recovery the iodide of potassium may be employed in moderate doses, continuously or intermittingly.

"An occasional week of mercurial may also be of benefit. Tonics are often called for, and among the best is cod-liver oil.

"The muscles may need massage or electricity. The patient should be made to sit up in a chair, and try to walk as soon as possible."

THE LOCALIZATION OF BRAIN DISEASE.—Prof. Henoch, in *Charitè Annalen*, fourth year, reports nine cases of tuberculosis of the brain, that show how risky it is to localize, basing this upon recent physiological investigations. The results are as follows:

SYMPTOMS.	LESION.
CASE I.—Left hemiplegia.	Multiple tubercles of the cortical layer of both hemispheres, the frontal lobes and tubercle of the left half of the cerebellum.
CASE II.—Tremor and paresis of the right side, finally, contraction of all extremities.	Tubercle of the left frontal lobe, the left corpus striatum, both thalami and right half of the cerebellum.
CASE III.—Hemiplegia and contracture of the left side, as well as of the facial nerve.	Tuberculosis of the right frontal lobe.
CASE IV.—Contracture and involuntary motion on right half of face and body.	Tuberculosis of the left frontal lobe.
CASE V.—Complete absence of symptoms until meningitis set in.	Tuberculosis of the commissure of the cerebellum and of both hemispheres.
CASE VI.—Paralysis of the left abducens, the left iris and right arm.	Tuberculosis of the commissure of the cerebellum.
CASE VII.—Absence until meningitis set in.	Tubercle in the pons.
CASE VIII.—Complete absence.	Tubercle of the left posterior lobe.
CASE IX.—Paralysis of the right abducens.	Tuberculosis of both posterior lobes, the posterior corpora quadrigemina, the pons and left crus cerebelli.

Of all these cases only II. and III. show the possibility that lesions of the motoric centre of the frontal convolutions produce motoric lesions of the opposite side. This chance of diagnosis, however, is very limited, as is shown by the other cases where these locations were free from disease, and yet the same symptoms produced with lesions in other parts of the brain, even cerebellum (Case VI.). Sometimes the intensity of the symptoms does not seem to correspond with the intensity of the lesion (V. and VI.). Henoch believes that a close study of the fibres leading from and to these physiological centres will do much to reconcile the apparent contradictions between pathological and symptomatological differences.—*Lancet and Clinic*, May 31.

INFLUENCE OF AGE, SEX AND MARRIAGE ON THE LIABILITY TO INSANITY.—In the *Journal of Mental Science*, for April, Dr. T. Algernon Chapman, from a detailed analysis of the statistics in the Report of the Commissioners in Lunacy, comes to the following conclusions:

1. These tables deal with sufficiently large numbers to give satisfactory results.

2. The numbers sent to asylums increase up to thirty-five years of age, when twelve per 10,000 living are annually sent; thereafter the number diminishes steadily to ten per 10,000 in old age. That if the age on first attack were given, instead of age on admission, and those always more or less congenitally defective were tabulated separately, the result would show a remarkably uniform proclivity to insanity throughout life, from thirty upwards, if not from twenty.

3. Insanity affects males more largely than females, from 20–40; again slightly more from 60 upwards; from 40–60, females are slightly prone. If general paralysis be treated separately, then females are much more affected from 40–60; at other ages there is an equality.

4. 3.5 per cent (1 in 30) of those who attain the age of 20 ultimately become inmates of asylums.

5. The single are sent to asylums in proportion greater than married, as 2.83–1; the widowed as 3–2, *i.e.*, in proportion to the numbers of each in the general population above 20 years of age, though the actual numbers of single and married admissions are nearly identical.

6. It is almost certain that in the excess of single above married, the excess is due, not to celibacy causing insanity, but to insanity or a tendency thereto preventing marriage. If this be so, about one per cent. of the single among the general population, aged 20–30, and about three per cent. of those aged 30–40, are so from mental defect ultimately causing their admission to an asylum.

7. General paralysis is more frequent among males than females; but at the age 40–50, when the disease is most frequent, this relative frequency is least marked.

8. Unlike insanity in the mass, general paralysis is hardly more frequent in the single than in the married, a circumstance probably traceable to the comparative rarity of congenital defect in general paralytics.

9. General paralysis results much more frequently than ordinary insanity from causes implying business energy, and the use (and abuse) of the activities of life; much less frequently from defects inherent in the individual.

SPASTIC SPINAL PARALYSIS (ERB); TABES SPASMODICA (CHARCOT).—In a paper read before the Berlin Medical Society and published in the *Berliner klinische Wochenschrift* for December 2d and 9th, 1878, Dr. Leyden says that he has observed a number of cases of which the clinical history, the necropsy, and the microscopic examination have been recorded, and which during life presented the symptoms of spastic paralysis. One case at a certain period of the disease presented strongly marked extension contracture and rigidity; but, later on as in the other case, there was flexion-contracture. All these cases, however, which had a fatal termination, presented also a high degree of paraplegia, partly associated with secondary atrophy of the paralyzed muscles, partly with disorders of sensation and of the bladder. *Post-mortem* examination showed chronic myelitis of various form and extent, affecting the dorsal portion, either alone or in conjunction with other parts of the spinal cord. He has also seen the symptoms of spastic paralysis presented in a number of other cases which did not end fatally, but in many of which an accurate anatomical diagnosis could be made. He classifies these cases under the following divisions:

1. Traumatic myelitis, in the later stages of which muscular spasms and contractures often occur. 2. Myelitis from compression of the spinal cord by carious vertebræ or by tumors. 3. Spontaneous myelitis. 4. Spinal paralysis, following acute disease, of which he has seen examples of the spastic form following typhus, small pox, and relapsing and intermittent fevers; in this category he also places puerperal paralysis. 5. Syphilitic paralysis, which, according to his observations, very commonly takes the form of spastic paraplegia; and 6, certain cases where the disease runs an acute or a subacute course, and extends upwards over the whole of the spinal cord. They may with great probability be regarded as meningitis or perimyelitis, and have a tendency to end in recovery. Dr. Leyden sums up his remarks in the following conclusions:

1. Spastic paralysis (characterized by rigidity of the muscles, muscular spasms, spinal epilepsy, increased tendon-reflex, extension and flexion-contractures) is a pretty frequent symptom in various diseases of the spinal cord; but it indicates no special form of disease. 2. It occurs in acute and subacute affections of the spinal cord, and in these circumstances is capable of perfect recovery. Cases of this kind are partly diffused (ascending) meningitis and perimyelitis (myelo-meningitis), partly circumscribed perimyelitis. 3. In acute myelitis (spontaneous, traumatic, or from compression), the symptom is, as a rule, not developed in the first stages of the disease, but very commonly appears in the second or chronic stage. In the same way, it is frequent in cases of myelitis with long-continued compression of the spinal cord. 4. The spastic form of paralysis is most frequently observed in chronic myelitis (sclerosis) when this occurs in one or more foci, one of which usually lies between the cervical and lumbar enlargements. In these cases, the periphery of the white anterior and lateral columns is more or less affected; there is, therefore, a varying extent of (chronic) leucomyelitis or myelo-meningitis. A special limitation of the sclerosis to the lateral columns has not yet been observed. 5. If it were desired to describe as a special form of disease those cases of chronic myelitis which are very slowly developed and remain free from disturbances of sensation and from paralysis of the bladder, they would correspond to the condition designated spasmodic tabes. But the expression "tabes," as Erb has correctly remarked, is applied in German literature to degeneration of the posterior columns; and the contrast of tabes atactica and tabes spasmodica would only be of importance, if the latter depended on a systematic sclerosis of the columns of the cord. In spite, however, of the remarkable symptoms these cases present no special type of disease either in anatomy or in symptoms. Ana-

tomically, they belong to chronic myelitis (leucomyelitis), the symptoms of which vary according to its stage and extent; symptomatically, neither the general commencement nor the absence of perceptible symptoms, is characteristic; for we see the same processes, at least the same anatomical lesions, after acute commencement, after repeated exacerbations, and when the course of the disease has been progressive. The absence of disturbance of sensation is accidental, and depends on the situation and extent of the focus of the disease Finally, in further progress of the case, the spastic symptoms may again disappear.—*British Medical Journal*, March 15, 1879.

THE CONDITION OF THE MUSCLES IN PARALYSIS.—In the paper above referred to, Dr. Leyden says, that spinal paralysis (and also cerebral and peripheral paralysis) may be divided into two great groups, according to the condition of the paralyzed muscles: *a.* Those in which the muscles are flabby, have lost their tonicity, and can be easily moved in all directions, so that the position of the limbs depends simply on gravitation; *b.* Those in which the tonicity of the muscles remains normal, and, as a rule, even increased, so that they have a tendency to contract and shorten and to oppose a tenacious resistance to extension. The first form occurs under conditions which diminish the tonus; the second under those which increase it. If we now concede to the sensory (or centripetal) nerve fibres an important influence over the muscular tonus, which we are justified in doing by the researches of L. Hermann and Cohnheim, and by Cyon's experiments on the influence of the posterior roots on irritability, the pathological facts become capable of a satisfactory explanation. Atonic paralysis is observed in the following conditions: 1. In extensive atrophy of the sensory fibres (posterior columns and nerve roots), in progressive locomotor ataxy. To this point Dr. Leyden has already directed attention in a monograph, published in 1863. In tabes dorsalis the muscles are flabby and atonic; but this does not exclude the possibility of the occasional occurrence of atonic condition. 2. Diseases of the grey matter interrupt the connection between the anterior and posterior roots by atrophy of the ganglion cells, without injuring sensation itself. It is hence conceivable that atrophic paralyses, proceeding from intense atrophy of the grey matter, belong to the atonic class. Slight diseases of the grey matter, especially of the interstitial tissue, are not unfrequently accompanied by a tonic condition of the muscles. 3. In cases where the irritability of the spinal cord, especially of the grey matter, is much diminished, atonic paralyses also occur. This is the case in the commencement of myelitic, and especially of traumatic affections of the portions of the spinal cord. Here we assume, with Goltz, an arrest of the function of the cord lying below the lesion, so that the reflex action is destroyed or greatly impaired, as also is the tonus of the muscles. This is the case in the last stage of severe diseases of the spinal cord, where the irritability, one might say the vitality, of the portion of muscle lying below the lesions, is destroyed, with, as a rule, general loss of strength. We here see atonic muscular paralysis appear, where, perhaps, distinct tonic paralysis has been present. This fact has not escaped the notice of Charcot, who says, that spastic symptoms may disappear towards the end of the case.

On the other hand, tonic or spastic muscular paralysis occurs under conditions which raise the reflex influence of the sensory fibres. This may occur: 1. From increased irritability of the motor fibres—neurotic peripheral paralysis. 2. From increased irritation of the sensory roots—neuritic or meningitic process. 3. From partial or complete interruption of the conduction of the will from the brain, the conduction of reflex impressions through the grey matter being preserved or even increased. Especially at the time when neuritic processes, often accompanied with much pain, are developed in the paralyzed limbs, muscular spasms and contractures begin to appear. What share the descending degeneration of the lateral columns has in this seems as yet doubtful. The condition is the same where there are myelitic foci, which intercept the conduction of the will from the brain, but leave intact the conveyance of reflex action through the grey matter (leucomyelitis).—*British Medical Journal*, March 15, 1879.

NEURITIS MIGRANS.—L. Putzel, M. D., *N. Y. Med. Record*, April 26, reports a case of neuritis of the internal cutaneous, external cutaneous, and ulnar nerves, from extension of inflammation from a boil over the right olecranon process. The neuritis extended upward along the ulnar nerve into the axilla and brachial plexus, being felt as a thick, indurated cord. Downwards it extended to the hand, causing paralysis of ulnar side of hand with anæsthesia and trophic changes. Evidences of inflammation also appeared in the auricularis magnus, producing redness of the tip of the ear and of a patch on the side of the neck, with anæsthesia. A similar patch also made its appearance below the inferior angle of the right scapula. Later in the disease, another similar patch appeared in the middle of the thigh anteriorly, and about six inches below Poupart's ligament; and still another over the left deltoid. The patient gradually improved under treatment and was lost sight of. All these, Dr. Putzel refers to the original neuritic trouble, and considers the case as clinically supporting the theory of neuritis migrans. The inflammation spread upwards along the ulnar nerve to the cord, producing there some medullary lesion, and then appeared again in the peripheral nerves (auricular, etc.). Just what this medullary lesion was cannot be told; it may have been a chronic disseminated myelitis, or the meninges alone may have been affected. The case though purely clinical is one of interest.

REFLEX INFLAMMATIONS.—Under this head have been described certain inflammations occurring at a distance, following a prolonged irritation of some part of the body, sometimes in a corresponding symmetrical locality, and always in a region innervated from the same region of the cord; it is in this manner that we explain sympathetic ophthalmia, and visceral lesions following extensive cutaneous burns, etc MM. Hallopeau and Neumann report in the *Gazette Médicale*, a case of inflammation explainable according to this theory. In a man troubled with rather indefinite thoracic pains, they applied two blisters successively to the same point, between the left border of the sternum and the nipple. There followed a severe pain with cutaneous inflammation involving the left nipple. These symptoms rapidly disappeared and the pain reappeared directly in the right breast, where there soon appeared pimples formed by the sebaceous glands of the part, many of these taking on the aspect of small boils; the whole breast became indurated, red, and painful, and from which abscesses formed, after opening which the whole inflammation disappeared. The authors consider this a case of reflex inflammation, it being produced not by propagation, as was easily seen, nor by mere coincidence, inflammations of the breast being exceedingly rare in the male. It may be said that the process seemed as if the irritation of the nerves appertaining to the left nipple and areola was transmitted to the spinal centre, and thence to the nucleus of origin of the nerves of the right areola, and through them provoked an irritation in that region. This is the mechanism called in by M. Vulpian to explain, in a general way, the production of reflex inflammations.—*Journal de Medicine et de Chirurgie Pratiques*, April.

MYSOPHOBIA.—At the meeting of the New York Neurological Society, April 7 (rep. in *N. Y. Medical Record*), Dr. W. A. Hammond read a paper upon the above subject, in which he described a form of mental derangement that consisted in a *fear of defilement or contamination.*

Ten cases had fallen under his observation, but not fully recognizing the exact nature of the earlier ones, he based his paper upon the complete clinical histories of the last three. In the first case described there was an overpowering desire to wash the hands, and in that occupation the patient spent a large share of her time. The fear of becoming contaminated gave her the most intense mental anxiety, suffering and distress; and although she was able to recognize the absurdity in her case, yet during her waking hours she was haunted and followed by what was to her a most terribly distressing fear.

In the second case the fear of pollution was more extended and serious, and the patient washed her hands as many as two hundred times a day.

The third case was equally well defined, but not so severe. When the patient visited the doctor's office she could not be induced to touch the door-knob when she was ready to leave the consultation-room, because of the tormenting and distressing fear of becoming contaminated, which held her in complete possession. The treatment which he had exhibited had been to keep the bowels quite soluble by means of pills composed of podophyllin, aloes, and ox-gall; to administer bromide of potassium, sodium, or calcium, and in combination with opium if there was a tendency to melancholia; and to use tonics—such as cod-liver oil, strychnia, iron, and quinine.

Dr. E. C. SEGUIN referred to a case in which the patient suffered through fear of croton bugs. The fear followed her, and she saw the bugs, when it was well established that there were none present. She was cured by moral treatment and by tonics.

Dr. SPITZKA spoke of mysophobia as a symptom which complicated different conditions, and

Dr. Kiernan mentioned cases of chronic mania in which he had seen similar symptoms.

Dr. Hammond remarked that the cases which he had reported, and to which he referred, were not cases of insanity, for they had neither hallucinations nor delusions.

This paper was subsequently published in full in Neurological Contributions.

Alcoholism of Parents as a Cause of Epilepsy in their Children.—Dr. Hippolyte Martin, *Ann. Méd. Psychol.*, Jan., gives an account of the investigations made by himself on the influence of intemperate habits of parents on their offspring as regards the production of epilepsy, while *interne* at the Salpêtrière. Out of one hundred and thirty to one hundred and fifty patients in the department for insane epileptics of that institution, he was able to obtain data as to heredity in eighty-three. Twelve of these cases are briefly detailed and an analysis of the whole is given. He divides them into two classes: in the first, comprising sixty patients or over two-thirds of the whole, intemperate habits in the parents was an established certainty; in the second class of these patients, the intemperance of the parents was dubious in some, and in others could not be suspected. The sixty of the first class had had two hundred and forty-four brothers and sisters; of this number forty-eight were affected with convulsions from early infancy, one hundred and thirty-two were dead in 1874, and one hundred and twelve only were still living, nearly all young and many of them with damaged nervous organizations.

The twenty-three epileptics of the second class had had eighty-three brothers and sisters, among whom only ten had had convulsions, and forty-six were still living. In these figures, of course, the patients themselves are not included. Of these eighty-three epileptics, all from different families, the particulars as to the origin of the disease could be obtained in seventy-eight, and it was found that fifty of them had first had eclamptic convulsions quite distinct from epilepsy. Thus in eighty-three families there had been four hundred and ten children. One hundred and eight of these, that is, more than one-fourth, had had convulsions; in 1874, one hundred and sixty-nine were dead, and two hundred and forty-one living, but of these latter, eighty-three, or more than a third, were epileptic.

In looking up the diseases of the parents and grandparents of these patients, the records of the following diseases were obtained:

Apoplexy	15
Heart Disease	5
Thoracic Disease	6
Suicides	4
Hysteria	5
Mental Alienation and Dementia	4
Cancer	7
Dropsy (?)	1
Chagrin (?)	1

It is not stated, but it is probable that the cause of death is here given in most but of course not all of these cases. The author remarks on the small proportion of diseases of the nervous system in the above, and hence concludes that to alcoholism alone must be attributed the epilepsy of the children. The large proportion of circulatory disorders (including under this apoplexy and heart troubles) which are directly favored by intemperance is also a striking fact. He concludes as follows: "We have therefore demonstrated that alcoholism in the ancestors is an extraordinarily frequent cause of eclampsia and epilepsy in their descendants; and if, as we have said in a former article, epileptics have only few children, and further, that these are almost inevitably affected by the most serious nervous disorders, we perceive what formidable evils alcoholism of the parents transmits to their posterity.

"Alcoholism is therefore the brutalization of the drinker and a miserable existence and speedy extinction of his posterity. This sombre picture may serve as an example to the unhappy victims of the appetite, and should, at all events, sustain the zeal and devotion of those who are striving to reform those beings whose degradation is at once a shame and a peril to the human species."

PATHOLOGY OF TETANUS AND HYDROPHOBIA.—At the meeting of the Pathological Society of London, April 29 (rep. in *British Medical Journal*), Dr. Ross showed microscopical sections illustrative of the pathology of the nervous system in hydrophobia and tetanus. He met with: 1, vascular engorgement along with evidences of effusion of a granular material and migration of leucocytes, described previously by Allbutt, Lockhart Clarke, Dickinson, Benedikt, and Gowers; 2, the yellow degeneration of the ganglion cells described by Allbutt; 3, in two cases of tetanus and two of hydrophobia, a large number of colloid bodies, and what appeared to be the condition described by Dr. Batty Tuke and by Mr. Kesteven as "miliary sclerosis." As, however, these cords were partially hardened in spirit, Dr. Ross was doubtful how far they might be regarded as evidences of disease, and he spoke with especial reserve as regarded the importance to be attached to the presence of colloid bodies. The most marked evidences of disease in the cord were found on each side of the central canal; but the posterior and anterior horns were affected. In tetanus, the vesicular column of Clarke was always injured, often completely destroyed; and it was also injured, but to a less extent, in hydrophobia. A distended vessel was generally observed to pass between the median and antero-lateral groups of ganglion cells of the anterior horns; and the ganglion cells on each side were destroyed, the destruction of cells being usually greater in tetanus than in hydrophobia, but never wanting. A similarly distended vessel was frequently observed passing through the postero-lateral groups, the destruction of ganglion cells here being usually more marked in hydrophobia than in tetanus.

In the inferior portion of the hypoglossal and spinal accessory nuclei, the cells were found nearly destroyed in tetanus; those of the hypoglossal were less so in hydrophobia; but the damage to the spinal accessory and vagus nuclei was much greater in hydrophobia than in tetanus. The condition of the medulla in hydrophobia corresponded closely to that described by Dr. Gowers. In two cases of tetanus, vascular changes and colloid bodies were found in the cortex and adjoining white substance of the cerebellum. In hydrophobia, colloid bodies, vascular changes, and at times yellow degeneration of the large cells of the fourth layer of the cortex, were observed. The condition described as miliary sclerosis was observed in two cases of hydrophobia. In the cord, the patches occupied the posterior and antero-lateral columns. Beyond the cord, the patches were observed in the anterior pyramids of the medulla and in their continuation through the pons varolii, middle third of the crusta, and middle third of the internal capsule. Similar patches were observed in the formatio reticularis and in the portion of the pons which corresponds to the anterior root-zones of the cord, and also in the parts of the corpus striatum adjoining the internal capsule. The state of the cord and medulla appeared, on the whole, to be very much the same in the two diseases, and the differences in the symptoms might perhaps be due to the fact that in tetanus the morbid changes were directed more to the cerebellum, while in hydrophobia the cerebrum was more affected.—Dr. Frederick Taylor said that he thought the presence of colloid bodies a very untrustworthy sign of disease, because they appeared to be produced by *post-mortem* changes in the cord. It was not possible to say exactly under what conditions they were developed, possibly by the action of spirit on cords in a certain stage of decomposition. Mr. Golding-Bird mentioned a case of chronic tetanus which had begun acutely, where the cord was found to be perfectly healthy in all parts. Dr. Ross, in reply, said that, though he did not wish to attach too much importance to the presence of colloid bodies, yet he could not altogether agree that they were always the result of *post-mortem* changes. He had seen them in cords which had been in neither spirit nor chromic acid. They were certainly not the result of changes in myeline matter, because he had found them in specimens of cheesy pneumonia.

PARALYSIS OF THE SERRATUS MAGNUS IN INFANTILE PARALYSIS.—At the meeting of the Clinical Society of London, May 23 (rep. in *Brit. Med. Journal*, June 7), Dr. D. B. Lees read notes of two cases of infantile paralysis which presented some peculiar features. We copy the report in the *British Med. Journal:*

Elizabeth M. was sent to the hospital for sick children; the muscles in worst condition were those of the left shoulder and right leg. In most of the muscles of these limbs faradic response was absent, and in many of the muscles of the left leg, and also in the right deltoid, it was much impaired. After the child had been brought to the hospital for a few weeks, it was noticed that there was marked falling in of the left side of the front of the chest during inspiration, instead of the normal expansion. The position of the left scapula was found to be altered; it was drawn up to a higher level than the right scapula, the upper angle was drawn nearer to the spinal column, and the lower angle had left the chest-wall and projected backwards. These symptoms seemed to justify a diagnosis of paralysis of the serratus magnus. On applying the electrodes of a battery over this muscle, a forward movement of the scapula at once responded to a current from seventeen Leclanché cells on the right side, but no movement could be obtained by the use of a much stronger current on the left. It was possible that in this case the interossei were also paralyzed, and certainly the rhomboidei had suffered, there being no muscular resistance when the scapula was pulled forwards. The child's general condition had since improved, but the position of the scapula and the electrical reactions were nearly unaltered. Herbert P., aged twelve months, was found completely paralyzed one morning early in December, 1878. When brought to the Hospital for Sick Children on December 20th, it was found that in him also the parts most deeply affected were the left shoulder and arm and the right leg. The right deltoid was decidedly paralyzed. It was observed that the angle of the left scapula projected from the chest-wall. On turning the child on his back, the left scapula fell away from the ribs. It was doubtful whether a less degree of a similar condition was not present on the right side also. Respiration was entirely diaphragmatic. There was no unilateral depression of the thoracic wall during inspiration. No response to electricity could be obtained in either serratus. This child had considerably improved under treatment. The right scapula was now in normal position, and the left nearly so. Some response to faradism had reappeared in both serrati and both deltoids had regained much power. He could now raise the hand above the level of the shoulder on the right side, but was still unable to do so on the left. In both these cases the serratus was only one of many muscles paralyzed, but the signs of its affection were distinct. Paralysis of this muscle was a rare condition, and Dr. Lees was not aware that attention had been previously drawn to it as an occasional complication of infantile paralysis.

Juvenile Insanity.—At the meeting of the Medical Society of Pennsylvania at Chester, May 21 (rep. in *N. Y. Med. Record*), Dr. I. N. Kerlin, Superintendent of the Home for Feeble-Minded Children, at Media, read a paper with the above title. Mental disease, he said, was more frequent in childhood than was commonly imagined. According to the statistics of Dr. Boutteville, the proportion of insane children, between the ages of five and nine years, was 10 per cent.; between ten and fourteen years, 35 per cent.; and between fifteen and nineteen years, 20 per cent. It was not improbable that many of the minor forms of juvenile insanity were allowed to go unrecognized, being regarded as a temporary consequence of sympathetic disturbance, or the sequel of acute disease, and so likely to pass away when the cause was removed, or overgrown. In many cases that happy sequence did not occur, and the little victims sank rapidly into the dementia of idiocy, or developed into erratic, excitable, vicious childhood, passing through the courts, and the refuges and the reformatories as criminals, from whence they graduated into other planes of crime.

REMISSIONS IN PRIMARY PARALYTIC DEMENTIA.—The following are the conclusions of a lengthy article by Baillarger in the *Annales Med. Psychologiques*, January, on remissions and dementia in certain cases of general paralysis, in which he analyzes some ten observations from various sources.

1. Melancholia with paralytic stupor or simple paralytic stupor may take on the most serious symptoms and simulate an advanced dementia, and nevertheless be followed, even after many months, by remarkable remissions.
2. When the symptoms of dementia, with some delirious ideas, develop rapidly, in the first period of general paralysis, they do not appertain to a genuine dementia, but to a *pseudo-dementia*, constituting a special condition not yet sufficiently studied.
3. It is often wrongly assumed that dementia exists from the beginning of general paralysis from certain special symptoms, or it is presumed to be masked by the maniacal or melancholic delirium.
4. The mobile, absurd and contradictory delirium of paralytics, is no proof of the existence of dementia, and may be explained by a special condition comparable to certain cases of intoxication.
5. The *pseudo-dementia* in general paralysis cannot as yet be distinguished from veritable dementia except by its rapid invasion or by signs of stupor.
6. There are no true remissions in the simple, slow and progressive paralytic dementia.

The following are the titles of some of the recently published papers on the Pathology of the Nervous System and Mind and Pathological Anatomy:

STILLMAN, Neurasthenia, *Med. and Surg. Rep.*, May 17.—THOMSON, on Astigmatism as a Cause for Persistent Headache and other Nervous Symptoms, *Med. News and Libr.*, June.—BEARD, Nervous Diseases Connected with the Male Genital Function, *N. Y. Med. Record*, June 14.—HAMILTON, On Epilepsy, *Ibid*, June 7.—MITCHELL, Spasmodic Disorders of Legs, *Ibid*, June 28.—BARTON, Dipsomania, Its Medical and Legal Aspects, *Practitioner*, June.—LAUDER-LINDSAY, Spurious Hydrophobia in the Lower Animals, *Edinb. Med. Journal*, June and July.—CLOUSTON, The Study of Mental Disease, *Ibid*, July.—BUMSTEAD, Sexual Hypochondriasis, *Am. Practitioner*, July.—GRAY, Cerebral Syphilis, *Proc. Med. Soc. Kings Co.*, July.—WRIGHT, Some Points Connected with the Question of Responsibility as it Relates to the Partially Insane, *Lancet and Clinic*, July 12.—MANN, The Psychological Nature and Significance of General Paralysis and its Pathology, *Detroit Lancet*, July.—NACKE, Delirium Tremens, *Centralbl. f. d. med. Wissensch.*, No. 25.—ROSENBACH, The Diagnostic Significance of the Reflexes, particularly the Abdominal Reflexes, *Centralbl. f. Nervenheilk.*, May 1.

c.—THERAPEUTICS OF THE NERVOUS SYSTEM AND MIND.

CHLORAL.—Dr. Geo. H. Savage, *Jour. of Mental Sci.*, April, discredits the valuation that has been placed upon chloral by many of the profession. He thinks it often misused, and that the weight of evidence is against it. He says: "I shall consider chloral, first as a possible cause of insanity, and next as a remedy.

"As a cause, I have seen its constant use for one or two years produce melancholia, associated with great prostration, loss of flesh and strength, a sallow, worn aspect, great irritability and nervousness, with strongly suicidal tendencies. I have the experience of several personal friends, men who were working hard with their brains, and who suffered from sleeplessness. These found chloral a boon at first, but by continuing its use they lost physical health, and the sleeplessness became more unbearable.

"It is rather difficult to explain this more active sleeplessness, but, doubtless, many of my readers know the less distressing stages of it. The chloral-taker often on going to bed, after a hard day's work, feels not only unable to sleep, but actively, restlessly wakeful, so that he is unable to remain more than a minute in one posture. There is no special or pleasing flow of thought, no equal brain activity, but often a feeling of depression, and a dreadful impulse to injure oneself without any object or cause of woe.

"This impulse was described rather as a feeling like the one impelling people to throw themselves from heights.

"In assuming that chloral was a cause of insanity, I must admit that often it was only one cause. The chloral-taker was sleepless from brain exhaustion, or brain irritation, from over-work or over-stimulation, and in some cases had insane inheritance; but this is true of many other causes. A shock or a blow may only produce insanity under similar circumstances. I have seen one case of insanity follow the suicidal taking of an enormous dose (two ounces) by a person used to excess of stimulants and the habitual use of chloral. This case became slowly weak-minded, and did not recover. In this case there was doubt expressed by at least one authority on the part played by chloral, as there were added other serious causes of trouble; but the fact remains that till the large and almost fatal dose was taken, marked insanity was absent.

"In several other cases the prolonged use of increasing doses of chloral seemed slowly to develop ill health and melancholia, accompanied by refusal to take food. In one the husband took a fatal dose, and his wife, who was also a chloral-taker, became insane.

"In another, an elderly woman exhibited, in an exaggerated form, the dread and agitation common to those using the drug, and had to be fed by the stomach-pump. She slowly lost strength in mind and body, and was discharged uncured.

"Though these are in no way peculiar in their form of insanity, I am convinced the cause was chloral. Of course, I do not believe that patients are made insane in asylums by the use of the drug, as the outside world has supposed, but I am convinced that many cases of acute insanity are injuriously affected, and, in some instances, allowed to die through the use of chloral. A large proportion of the cases of puerperal insanity that are now admitted into Bethlehem have passed through a course of chloral treatment; which means that they have been dosed into quietness, till they have refused food. Such cases take longer in recovering, and are more troublesome to treat, from their physical exhaustion and their refusing food.

"Having, so far, only expressed my dislike to the drug, or, in other words, spoken of its abuse, I would pass on to say what I believe to be its uses.

"It has been used in—1st, sleeplessness; 2d, in various forms of insanity; 3d, in several stages of epilepsy. In reference to the first, I have seen sleep produced by chloral, when the wakefulness was not due to pain; but if this sleeplessness were the initial stage of insanity, I have not seen the attack warded off by the sleep. In a few cases, where over-work or anxiety is affecting the health, a single dose of twenty-five grains at bedtime is of service, but I would rarely give it regularly.

"For simple sleeplessness in the brain-worker, who has little or no bodily exercise, sleep may be induced, and work got through that could not have been done without some sleep; but, in my experience, this is done by drawing on the capital of energy, and, if pursued, will end disastrously.

"In a few sleepless persons, chloral seems to act uniformly well, and does not require increasing doses, and does not affect the appetite or general functions. In such cases it may be of great service.

"I believe, however, that simple sleeplessness is much better treated by other general measures before chloral is tried.

"2d. In some cases of maniacal excitement, the patients are controlled and made manageable. According to some authorities, the sleep induced in some of the most violent of such cases saves them from death by exhaustion. This seems probable; but in practice I find stimulants and abundant light food act just as well.

"In some cases of recurrent mania with great violence the drug has been given in large and repeated doses, gradually increasing till at length we have reached two drachms every four hours, and no beneficial result has followed. In melancholia one does not get good results by either occasional or regular administration.

"In general paralysis I have not dared to give the drug any long trial, as I felt that I was running the danger of removing one stone from the tottering structure.

"In cases of insanity following blows, shocks, and the like, I believe excitement is produced much like that produced by alcohol in cases of injury to the head.

"In acute insanity with intemperance, a few cases were relieved by chloral.

"In speaking above of the treatment, I refer to my experience of chloral alone. In some future papers I may speak of its combination with other drugs.

"In the third group I place epileptics; and with these I have greater faith but less experience.

"I know of no good results following the use of the drug in simple epilepsy; but in patients who have furor associated with the fits, the benefit is very great and very certain.

"I have frequently seen a case that without chloral was dangerously maniacal, for several days sleep quietly, after thirty grains of chloral, and wake up sane, having had an epileptic fit, but no furor. Every fit that was not followed by a dose of chloral, was followed by a fit of mania.

"I have now expressed my belief, which may be summed up to be that chloral may produce physical ill-health, hypochondriasis and insanity. It may relieve epileptic furor, but cannot cure epilepsy; it may produce sleep in some cases with advantage, but more commonly disadvantageously; it may be used as restraint rather than as treatment in violent cases."

CODEIA.—Dr. Robert Saundby (*Brit. Med. Jour.*, April 12,) recommends codeia as a sedative against frequent coughing in patients in whom for various reasons opium and morphia are contra-indicated. He claims that in phthisis, it allays cough without deranging digestion, and that it also agrees with gouty individuals in whom morphia and opium are not tolerated. Its value in diabetes is of course fully recognized. He prescribes it in doses up to one grain in syrup of tolu.

RESULTS OF TREATMENT OF THE INSANE.—At the meeting of the Medical and Chirurgical Faculty of Maryland, April 10, Dr. J. S. Conrad, chairman of the Section on Psychology, read a paper on the Treatment of the Insane, the following abstract of which we take from the report of the proceedings in the *Maryland Medical Journal* for May.

Dr. Conrad began his paper by remarking that the modern expensive and palatial hospitals for the insane have been the outcome of asylum reports, showing a very large percentage of recoveries, ranging from 60 to 90 and, in one case, 100 per cent. Whilst compared with these exhibits the almshouse care and treatment was shown to realize only seven per cent. of recoveries. The data upon which such reputed success has been claimed have been shown by indisputable evidence to be fallacious and untruthful, and that the error of the asylum reports consists in counting the cases recovered and not the *persons.* Thus Dr. Pliny Earle, superintendent of a Massachusetts asylum, has enumerated instances of this method of computing the ratio of recoveries.

One man was discharged recovered seven times in nine years, and afterwards committed suicide; another was discharged 14 times, another 26 times, and again a woman was admitted 59 times, and discharged cured, in 29 years, and finally died insane. These examples are cited to show the fallacy of asylum reports.

Dr. Conrad remarked that the ratio of recoveries had become the standard of judgment of an institution; as a consequence such a rivalry in the ambition of superintendents to show the greatest number of recoveries. This was the result of the reaction from the old almshouse treatment, or no treatment at all.

The reports of these large percentages of cures have gradually dwindled down for several years past under the severe criticism of Dr. Earle, and the actual results are illustrated by the yearly accumulation of chronic cases. This reaction is still continuing as the pruning knife of truth is applied.

Thus Dr. Skae, inspector of the insane for the colony of New Zealand, in a report shows the results of the almshouse and "wild beast method" of treatment performed in New Zealand, in contrast with the English, Irish and Scotch county and borough asylums. Notwithstanding the deplorable manner in which the insane are treated in this colony, Dr. Skae says the ratio of recoveries to admissions is 13 per cent. higher than in the Scotch and Irish, and 23 per cent. higher than in England; their death-rate 4.4 per cent., being 4.5 per cent. lower than in England.

Dr. Conrad from the report of Dr. Skae was led to make comparisons of our own system, and to that end reviews the reports of the City Almshouse, Spring Grove Asylum, and Mount Hope Retreat for the Insane. The comparative results of the institutions are equally surprising as that reported by Dr. Skae. The following table of results of figures taken from the last published reports of these respective institutions as prepared by Dr. Conrad are here given:

	BAY VIEW.	SPRING GROVE.	MT. HOPE.
Cost of maintenance, per capita	$69.37	$214	Not given.
Number of Insane treated	266	434	445
“ “ admitted	86	153	131
No. of Insane recovered and improved	77	94	72
Per cent. of same on admission	per cent. 89.5	61.	41.
“ “ “ the whole	“ 28.9	21.4	16.2
Whole number of deaths	12	34	29
Per cent. of deaths	4.5	7.8	6.5

These results in favor of the Bay View Asylum (or Almshouse), are more remarkable when it is noted that the physicians of this institution state in their report (page 44) "that we are only permitted to receive or to retain idiots, imbeciles, epileptics, and chronics ; all acute and curable cases are sent to Spring Grove and other special institutions."

Dr. Conrad next shows the sanitary condition of these institutions, and the methods of treatment employed. He presents the report of Dr. Chancelor, on public charities, reformatories, prisons and almshouses, 1877, to illustrate the surroundings and poor comforts of the City Almshouse in contrast with the elegance and beauty of Spring Grove Asylum. In conclusion, Dr. Conrad remarks, "but for the report of Dr. Skae showing a similar comparative picture in New Zealand, he would be unable to account for such statistics except upon the ground that the report from Bay View was incorrect."

SECTION OF THE TRIGEMINUS FOR THE CURE OF TIC DOULOUREUX.—Dr. F. S. Dennis, (*N. Y. Med. Jour.*, June,) after reviewing some twenty odd cases of resection of the superior maxillary nerve which he has collected, sums up as follows:

1. The high operation upon the superior maxillary nerve for the cure of neuralgia characterized by pain (loci dolentes, *points douloureux*), having its maximum of intensity at the infra-orbital foramen, in the course of the malar branch, and sometimes, though rarely, in the alveolar dental, superior labial, and palatine points, is a better operation than the lower one, inasmuch as the preceding cases show that the former, as a secondary measure, has cured or brought relief in nearly every case in which the latter has failed to do so.

2. The operation is justifiable, as the record shows no case in which death has occurred as an effect of the operation, nor one in which the symptoms have been aggravated.

3. Relief, if only temporary, is a result which justifies the high operation for neuralgia as much as it does the one for the removal of malignant tumors, which, as a rule, invariably recur subsequently to their extirpation by the knife.

4. Want of success is due to failure as regards a correct diagnosis, not only of the cause of the pain, but also to failure as regards the site of the disease. If central, the diagnosis fails to determine whether the centres are primarily or secondarily involved, or, if peripheral, whether the recurrent fibres are or are not implicated.

5. Failure will be the rule rather than the exception, unless care is taken to distinguish those cases in which neuralgia is due to central lesions from those in which the cause lies in the periphery.

6. If the lesion is situated in the terminal nerve plexuses, polyneurotomy, or the resection of several nerves, will be more likely to remove pain than mononeurotomy, or the resection of a single nerve.

7. Mononeurotomy should be reserved for those cases in which the irritant involves the trunk of a nerve near its origin.

8. The prognosis will be the most favorable where lesions involving the plexuses or the nerve centres can be excluded.

9. The principal indication for the employment of this bold but safe operation is found in the obstinacy and persistency of pain, notwithstanding the trial of all milder measures.

Finally, when the case is a desperate one and as a *dernier ressort*, every conscientious and skillful surgeon should not fail to make a trial of an operation which facts prove to be as good in theory as it is successful in practice.

In conclusion, it may be said that resection of the superior maxillary nerve, together with Meckel's ganglion, for the alleviation, and in most cases the cure, of one of the most dreadful and formidable diseases to which human flesh is heir, is one of the greatest triumphs of surgery. A careful analysis of the table will show that the two cases belonging to Wood and one to Schuppert remained free from pain at least two years; and the interesting and unique autopsy held in the case of Mike Doyle, one of the severest cases on record, has thrown much light upon the subject of accuracy in diagnosis, upon the pathological changes involved, and upon a right estimate of untold value of this most daring operation. It is also a most significant fact that out of the twenty-one cases that have been collected from all the literature upon this subject more than half have been performed by American surgeons. It is certainly an operation of which American surgery can justly be proud; and if the success which has attended these twenty-one cases will only induce surgeons to investigate impartially the merits of this operation, it will inaugurate a new era and establish a reform in the surgical treatment of this most desperate malady.

METALLOSCOPY.—The following is the *resumé* of a thesis recently sustained at the Frederick William University, Berlin, by A. S. Adler, a Californian student there, as given by M. Blanchard in the *Progrès Médical*, May 3:

The author first gives succinctly the state of the question of metalloscopy, then, in a following chapter he relates his own experiments, and the whole is summed up in a final chapter that I translate *verbatim*:

"In cases of hysterical hemianæsthesia, a simple excitation, such, for example as that from a mustard poultice, if kept up long enough, produces under any circumstances an increase of sensibility of the part affected. This increase may sometimes have only the effect to recall the lost sensibility; but it may even provoke such a hyperæsthesia as to make simple contact painful. This restoration often coincides with a diminution of sensibility at a point symmetrical to the part excited, but this result is not a constant one. With the use of metals less constant results are obtained in this order of observations, than with mustard plasters. Of four metals tested on a hemianæsthetic patient, only copper caused any augmentation of sensibility in the part to which it was applied; an increase sometimes coincident, sometimes not, with a corresponding diminution in a symmetrical portion of the body.

"In healthy individuals, the application of metals gives very inconstant results; sometimes they increase sensibility of the point to which they are applied; at other times, on the contrary, they decrease it; and again in others they produce no effect whatever. We cannot therefore speak of any certain influence of metals exerted on the sensibility of the healthy individual.

"I have, on the other hand, seen regularly and surely, that a simple excitation (sinapism) increases the sensibility of the point of application and diminishes that of a symmetrical point on the other side of the body. Hence it follows that, when the metals exert an influence on the sensibility of hysterical patients, this may be explained very probably, in an altogether different way than by supposing that they have a specific action on sensibility.

"The important result of my investigations is the fact that, in the healthy individual, a simple excitation increases the sensibility of the side not excited (healthy) and diminishes that of the side excited (diseased). It is beyond a doubt that this phenomenon appertains to the functions discovered by Adamkiewicz, and described by him under the name of 'bilateral functions.' If in one class of these functions (the secretion of sweat) we have the synergic action of centres of organs symmetrically situated, the sensibility of the body represents on the other hand a bilateral function in which the ganglia of symmetrical organs are antagonistic to each other in the exercise of their functions."

THE THERAPEUTIC VALUE OF CROTON CHLORAL.—In a very interesting paper read before the Ulster Medical Society, Dr. Riddell (*Dublin Medical Journal*, April, 1879) reports his experience of the great therapeutical value of croton (butyl) chloral. He mentions first a case of severe paroxysmal headache ineffectually treated for many years by all the great guns of the *Pharmacopœia*, but cured by five grains of butyl-wine and glycerine, with a little acid and syrup of orange to cover the flavor. The patient continues the five-grain doses at night, and now enjoys better health than she has done for years. Since that case, Dr. Riddell says, he has used it largely—sometimes failing, sometimes relieving—till, by keeping an account of all his cases, it began to be clear which were most benefited by the drug. Since then, the number of cases relieved (some permanently) has increased. These cases are : headache in females arising from mental distress ; those cases of headache frequent at the menopause—in fact, all those called neuralgic except a few arising from internal mischief, are benefited, and in many instances cured. In that distressing species of neuralgia called tic doulourcux, he has found it in many cases acting like a charm. Of course, he does not include any arising from cranial or inter-cranial causes. He has tried it in neuralgia of the ovaries, but no good resulted. In insomnia, it is not so reliable as the hydrate ; but in some cases, where the loss of, or inability to, sleep is accompanied by a weak or fatty heart, it is to be preferred, as it has no weakening effect on the central organ of the circulation. In one case of delirium tremens, where the circulation was very feeble, the combination of croton chloral with digitalis had a wonderful effect, and it seemed as if the drugs could be given together in much smaller doses to produce the same results than singly. In this he pushed it from ten to thirty grains every three hours, with drachm and two-drachm doses of the infusion of digitalis. In pain arising from caries of the teeth, he has found it useless in most cases, and in all inferior to Richardson's "tinctura gelsemini ;" but in one case, of a nervous young lady, by giving her two ten-grain doses, he was able to extract a tooth next to painless to her great satisfaction. In these cases it is in affections of those parts supplied by the fifth pair of nerves that it is of most use ; but, to be of service, the drug must be given in far larger doses than prescribed in the *Pharmacopœia* for adults, five grains three or four times daily, gradually increasing if required ; if stimulants be wanted, dissolve it in rectified spirit ; if not, dissolve it in glycerine. In all cases complicated with hæmorrhoids, give glycerine. If anæmia exists, combine it with iron, or what he believes better, arsenic ; then gradually lessen the chloral. In all cases he has found it better to give it in solution than in powder or pill. Dr. Riddell mentions also severe pain with photophobia and blepharospasm after injury, in which atropia failed, but ten grains of butyl-chloral repeated in an hour gave complete relief; and a case of acute painful facial carbuncle, in which the effect of ten-grain doses every three hours was "simply marvelous," the disease going through its subsequent stages almost without the patient knowing anything of the matter from the sense of feeling. This remedy is probably less used in practice than its remarkable anodyne powers deserve.—*Brit. Med. Jour.*, May 3.

THEVETIN.—The following are the conclusions arrived at from an experimental investigation of the glucoside of *Thevetia iccotli* (Yoyote, yellow narcissus), a Mexican plant, by David Cerna, M. D., and published in the *Philadelphia Medical Times*, May 24 and June 7. The experiments were performed on dogs, cats, mice, rabbits, guinea pigs and frogs:

I. Thevetin is a very powerful poison, the minimum fatal dose in the common frog (Rana esculenta) being $\frac{1}{60}$ of a centigramme.

II. Thevetin produces death by asphyxia and by cardiac paralysis, more frequently by the former.

III. Thevetin is an irritant when locally applied to the skin, giving rise to a peculiar burning sensation.

IV. The diminution in the number of heart pulsations produced by thevetin is due to its action on the cardiac muscles. The poison seems to have no action on the pneumogastrics.

V. The arterial pressure is increased by an action of the drug on the heart itself, *i. e.*, by a stimulating action on the intra-cardiac ganglia.

VI. The primary acceleration in the number of respirations produced by thevetin is due to excitation of the centre in the medulla oblongata, the subsequent decrease and the final cessation of the respiratory movements being due to an action on the functional nerves, and also, probably, to an action on the muscles of respiration particularly.

VII. The convulsions produced by thevetin are cerebral.

VIII. The paralysis produced by the poison is spinal.

IX. As sensation is lost before voluntary movements, and as the nerves remain intact after death, it is evident that the abolition of reflex activity is of spinal origin, and is dependent upon an action of the drug on the sensory tract of the cord.

X. Thevetin increases intestinal peristalsis.

XI. Like most other drugs, thevetin lowers the temperature.

XII. When locally applied, thevetin produces contraction of the pupils, due to peripheral irritation.

XIII. In thevetin-poisoning the salivary is the only secretion markedly increased.

THE ACTION OF ANÆSTHETICS.—The *British Medical Journal*, June 21, contains the third report on the action of anæsthetics made to the Scientific Grants Committee of the British Medical Association, by Drs. Coats, Ramsay, and McKendrick. They conclude their reports as follows:

1. Both chloroform and ethidene administered to animals have a decided effect in reducing the blood-pressure, while ether has no appreciable effect of this kind.

2. Chloroform reduces the pressure much more rapidly and to a greater extent than ethidene.

3. Chloroform has sometimes an unexpected and apparently capricious effect on the heart's action, the pressure being reduced with great rapidity almost to *nil*, while the pulsations are greatly retarded or even stopped.

The occurrence of these sudden and unlooked-for effects on the heart's action seems to be a source of serious danger, all the more that in two instances they occurred more than a minute after chloroform had ceased to be administered and after the recovery of the blood-pressure.

4. Ethidene reduces the blood-pressure by regular gradations, and not, so far as observed, by sudden and unexpected depressions.

5. Chloroform may cause death in dogs by primarily paralyzing either the heart or the respiration. The variations in this respect seem to depend to some extent on individual peculiarities of the animals: in some, the cardiac centres are more readily affected, in others, the respiratory. But peculiarities in the condition of the same animal very probably have some effect in determining the vulnerability of these two centres respectively, and they may both fail simultaneously.

6. In most cases, respiration stops before the heart's action; but there was one instance in which respiration continued when the heart had stopped, and only failed a considerable number of seconds after the heart had resumed.

7. The use of artificial respiration was very effective in restoring animals in danger of dying from the influence of chloroform. In one instance its prolonged use produced recovery, even when the heart had ceased beating for a considerable time.

8. Under the use of ethidene, there was on no single occasion an absolute cessation either of the heart's action or of respiration, although they were sometimes very much reduced.

It can, therefore, be said that, though not free from danger on the side of the heart and respiration, this agent is in a very high degree safer than chloroform.

9. These results confirm and amplify those stated in a previous report, to the effect that ethidene does not compromise the heart as does chloroform. By the method of experimentation then employed, the effect on the blood-pressure could not be determined; and altogether the results here obtained are more exact and unequivocal.

It may be added that since the last report, ethidene has been given to a number of patients of all ages, with results which may be described as satisfactory.

Given freely at first, it produced anæsthesia as rapidly as chloroform, and the effect could readily be kept up by comparatively small subsequent doses. The only drawback is that in some cases it produced vomiting; but it has not been determined that it does so more frequently than chloroform, over which it has the further advantage of producing less excitement and being more agreeable to the patients.

Isobutyl chloride was given to three patients, but it produced considerable excitement, and proved an imperfect anæsthetic.

In concluding this report, the committee would express the belief that, so far as purely physiological tests go, ethidene has proved itself as efficient an anæsthetic as chloroform, and a much safer one. It is not asserted that this agent ought at once to replace chloroform in practice; but it is believed that a very strong case has been made out for an extensive trial of it. It is impossible for the committee, having numerous other engagements, to give the agent that extended trial in actual practice which is desirable; and they would ask those who are engaged in practice to use it in their cases, and to report the results they may have obtained.

It appears that ethidene was first employed as an anæsthetic by Dr. Snow. He administered it in fifteen cases, with good results (see *Snow on Chloroform*, etc., last paper, published in 1858). In 1870 it was used by Liebreich and Langenbeck in Berlin (*Berlin klin. Wochenschrift*, Nos. 31 and 33 1870, p. 401). In 1871, two papers appeared: one by Sauer, in the *Pharm. Centralblatt*, vol. XIV., p. 140; and the other by Steffen, in *Deutsche Klinik*, No. 44, p. 398. Sauer mentions one case of death in a patient suffering from heart disease. In thirty-three cases, two vomited, and two suffered from nausea and headache. In 1872, Steffen publishes another paper in the same journal (p. 358), in which he gives details regarding twenty cases; and he states that the results were satisfactory. (See, also, *Jahrb. der Medicin*, 1870, 1871 and 1872, where abstracts are given.)

Snow states that the difficulty in obtaining ethidene pure may prevent its general use. That difficulty is now removed; and there is little doubt that, if required, ethidene may be made in a state of purity and at a moderate cost.

THE following are a few of the recently-published papers on the Therapeutics of the Nervous System and Mind :

WARNER, Jaborandi in the Treatment of Puerperal Albuminuria and Convulsions, *N. Y. Med. Rec.*, July 12.—COGHILL, Nitrite of Amyl in Chloral Poisoning, *Brit. Med. Jour.*, June 28.—GAUSTER, The Cure of General Paralysis, *Jahrbücher f. Psychiatrie*, I., 1879.—SAVAGE, Hyoscyamin and its Uses, *Jour. of Mental Science*, July.—MATTISON, A New Method of Treatment of Opium Inebriety, *Quart. Jour. of Inebriety*, June.—BERGER, The Electric Treatment of Convulsive Tic and Chorea Minor, *Centralbl. f. Nervenheilk.*, May 15.—HAMMOND, The Non-Asylum Treatment of the Insane, *Neurol. Contributions*, I.

BOOKS, ETC., RECEIVED.

Pathologie der Bevölkerung. Studien über menschliche Gebrechen und deren Bedeutung für das Leben des Einzelnen und der Gesellschaft. Von Edward Reich. Berlin. 400 pages.

Zur Allgemeinen Physiologie der irritabeln Substanzen. Von Dr. B. Luchsinger. Bonn. 1879.

Die Einheit der Wissenschaft. Aus dem Gesichtspunkt der Lehre vom Leben. Von Jac. Moleschott. Giessen. 1879.

Considerations sur la Sensibilité dans ses Rapports Généraux avec les Phenomenes Psychiques. Par le Docteur A. E. Mordret. Paris. 1879.

A Clinical Treatise on Diseases of the Liver. By Dr. Fried. Theod. Frerichs. In three volumes. Vols. II. and III. Translated by Chas. Murchison, M. D., F. R. C. P. Wm. Wood & Co. New York. 1879.

Diseases of the Intestines and Peritoneum. By John S. Bristowe, M. D., J. R. Wardell, M. D., J. W. Begbie, M. D., S. O. Habershon, M. D., T. B. Curling, F. R. S., and W. H. Ransom, M. D. Wm. Wood & Co. New York. 1879.

Electricity as Related to Medicine and Surgery. By A. D. Rockwell, A. M. Wm. Wood & Co. New York. 1879.

Studié di Termometria Cerebrales Negli Alieniati. Pei Dottori D. Maragliano, E. G. Seppilli. (Estratto Dalla *Rivista Sperimentale di Freniatria e de Medicina Legale.*)

Other Symptoms of Nervous Exhaustion (Neurasthenia). By George M. Beard, A. M., M. D. (Reprinted from the JOURNAL OF NERVOUS AND MENTAL DISEASE, April, 1879.

American Nervousness, its Philosophy and Treatment. By George M. Beard. (Reprinted from *Virginia Med. Monthly*, July, 1879.)

Pott's Disease, its Pathology and Mechanical Treatment, with Remarks on Rotary Lateral Curvature. By Newton M. Shaffer, M. D. Jansen, McClurg & Co., Chicago. G. P. Putnam's Sons, New York. 1879.

Laryngeal Tumors and Tuberculous Laryngitis. By E. F. Ingals, A. M., M. D. (Reprinted from the *Chicago Medical Journal and Examiner* for July, 1879.

On Some Points in Connection with the Treatment of Sterility. By A. Reeves Jackson, A. M., M. D., Chicago. (Reprint from Vol. III., Gynecological Transactions, 1879.)

Fifth Biennial Report of the Board of State Commissioners of Public Charities of the State of Illinois. November, 1878.

Third Annual Report of the State Board of Health of the State of Wisconsin for the Year ending December 31, 1878.

Sixteenth Annual Report of the New York Society for the Relief of the Ruptured and Crippled. May, 1879.

The Utility of the Sphygmograph in Medicine. By T. A. McBride, M. D. (Reprinted from *Archives of Medicine*, April, 1879.) G. P. Putnam's Sons, New York. 1879.

Perineorrhaphy, with Special Reference to its Benefits in Slight Lacerations, and a Description of a New Mode of Operating. By Edward W. Jenks, M. D. (Reprinted from the *American Journal of Obstetrics*, April, 1879.) Wm. Wood & Co., New York. 1879.

Urethrismus, or Chronic Spasmodic Stricture. By F. N. Otis, M. D. (Reprinted from *The Hospital Gazette*, April 19, 1879.)

Inhalations in the Treatment of Pulmonary Diseases. By F. H. Davis, M. D., Chicago. (Reprinted from *Detroit Lancet*, May, 1879.)

Posture, as a Means of Relief in Strangulated and Incarcerated Hernia, with a General Consideration of the Mechanism of Reductions. By Frank H. Hamilton, A. M , M. D. (Reprinted from *The Hospital Gazette*, June 7, 1879.)

The Radical Cure of Hernia by the Antiseptic Use of Carbolized Cat-gut Ligature. By Henry O. Marcy, A. M., M. D. (Reprinted from the Transactions of the American Medical Association, 1878.)

Proposed Legislation on the Adulteration of Food and Medicine. By Edward R. Squibb, of Brooklyn. (Reprinted from the Transactions of the Medical Society of the State of New York for 1879.) G. P. Putnam's Sons. 1879.

On the Permanent Removal of Hair by Electrolysis. By George H. Fox, A. M., M. D. (Reprinted from the *Medical Record*, March 22, 1879.)

Mediums and their Dupes: an Explanation of So-called Spiritual Phenomena. Sydney, Australia. 1879.

Finger Stumps. By H. C. Fairbrother, M. D. St. Louis. (Reprinted from the *St. Louis Medical and Surgical Journal*, May, 1879.)

Posological Table. Including all the Officinal and the Most Frequently employed Unofficinal Preparations. By Charles Rice. Wm. Wood & Co., New York. 1879.

Opium as a Tonic and Alterative, and its Hypodermic Use in the Debility and Amaurosis sometimes consequent upon Onanism. By B. A. Pope, M. D. (Reprint from the February number of the *New Orleans Medical and Surgical Journal.*)

Chloral Inebriety. By J. B. Mattison, M. D. (Read before the Kings County Med. Society, Brooklyn, N. Y., April 15, 1879.)

The Microscope in its Relation to Medicine and Cerebral Pathology. By J. N. De Hart, M. D. (Reprint from the *Chicago Med. Journal and Examiner* for March, 1879.)

Constitution and the list of Members of the American Public Health Association. New York. 1879.

Forty-seventh Annual Announcement of the Faculty of Medicine of McGill University. Montreal. 1879.

Proceedings of the Sanitary Council of the Mississippi Valley. Memphis, April 30, May 1 ; Atlanta, May 5–9.

The Fifty-fifth Annual Report of the Officers of the Retreat for the Insane at Hartford, Conn. April, 1879.

Thirty-third Annual Announcement of Starling Medical College for the Session of 1879–80. Columbus, Ohio.

Saratoga as a Remedial Watering Place. By L. E. Whiting, M. D. (From the *N. Y. Medical Record.*)

THE FOLLOWING FOREIGN PERIODICALS HAVE BEEN RECEIVED SINCE OUR LAST ISSUE.

Allgemeine Zeitschrift fuer Psychiatrie und Psychisch. Gerichtl. Medicin.
Annales Médico-Psychologiques.
Archiv fuer Anatomie, Physiologie, und Wissenschaftl. Medicin.
Archiv fuer Path. Anatomie, Physiologie, und fuer Klin. Medicin.
Archiv fuer die Gesammte Physiologie der Menschen und Thiere.
Archiv f. Psychiatrie u. Nervenkrankheiten.
Brain.
British Medical Journal.
Bulletin Générale de Thérapeutique.
Centralblatt f. d. Med. Wissenschaften.
Centralblatt f. d. Nervenheilk., Psychiatrie, etc.
Cronica Med. Quirurg. de la Habana.
Dublin Journal of Medicine and Surgery.
Deutsche Medicinische Wochenschrift.
Edinburgh Medical Journal.
Gazetta Frenocomia di Reggio.
Gazetta Medica de Roma.
Gazette des Hopitaux.
Gazette Médicale de Bordeaux.
Glasgow Medical Journal.
Hygeia.
Hospitals Tidende.
Journal de Médicine et de Chirurgie Pratiques.
Journal of Mental Science.
Journal of Physiology.
Journal de Medecine de Bordeaux.
La France Médicale.
Lancet.
Le Progrès Medical.
Lo Sperimentale.
L'Union Medicale.
Mind.
Nordiskt Medicinskt Arkiv.
Norsk Magazin for Lagensvidenskabens.
Psychiatrisches Centralblatt.
Rivista Clinica di Bologna.
Rivista Sperimentale di Freniatria e de Medicina Legale.
Revue Medicale du Nord-Est
Revue Mensuelle de Medicine et de Chirurgie.

Schmidt's Jahrbuecher der In- und Auslandischen Gesammten Medicin.
St. Petersburger Med. Wochenschrift.
The Practitioner.
Upsala Lakareforníngs Forehandlinger.

The following domestic exchanges have been received:

American Journal of Insanity.
American Journal of Medical Sciences.
American Journal of Obstetrics.
American Journal of Pharmacy.
American Medical Weekly.
American Practitioner.
Archives of Dermatology.
Atlanta Medical and Surgical Journal.
Boston Medical and Surgical Journal.
Buffalo Medical Journal.
Bulletin National Board of Health.
Bulletin of Medico-Legal Society.
Canada Medical Record.
Canadian Journal of Medical Sciences.
Chicago Medical Journal and Examiner.
Cincinnati Lancet and Clinic.
Detroit Lancet.
Index Medicus.
Maryland Medical Journal.
Medical Brief.
Medical News and Library.
Medical Record.
Medical and Surgical Reporter.
Michigan Medical News.
Nashville Journal of Medicine.
New Remedies.
New York Medical Journal.
New York Med. and Surg. Brief.
Ohio Medical and Surgical Journal.
Pacific Medical and Surgical Journal.
Proceedings of Med. Society of the County of Kings, N. Y.
Pharmacist and Chemist.
Philadelphia Medical Times.
Quarterly Journal of Inebriety.
Richmond and Louisville Medical Journal.
Southern Practitioner.
Southern Clinic.
St. Louis Courier of Medicine.
St. Louis Medical and Surgical Journal.
St. Louis Clinical Record.
Toledo Medical Journal.
Virginia Medical Monthly.

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Original Articles, Selections and Translations.

Art. I.—THE CREMASTER-REFLEX.

By S. Weir Mitchell, M. D.
Member of the National Academy of Sciences.

IN 1864 my attention was first called to the reflex movements of the cremaster muscles by a case in which a bullet wound of the inside of the thigh and of the sciatic nerve, caused retraction of the testicle for several hours.

In 1872, in my work on "Nerve Injuries," p. 137, I mentioned this case and briefly described the cremaster-reflex and the region of skin capable of producing it.

Within a year I have more carefully re-studied the subject with the hope of finding new and useful diagnostic help in cases of spinal disease. I have been disappointed in attaining valuable results in this direction, but I have learned enough that is curious and physiologically interesting to make it worth while to state with brevity the results of my observations.

The muscle slips which constitute the cremaster are portions of the lower edge of the belly of the internal oblique muscle and transversalis. The reflex arc arises on one hand in the skin of a large part of the thigh by twigs of the anterior crural, internal cutaneous, middle cutaneous, two saphenous, and anterior twig of obturator.

Through the lumbar enlargement of the cord the arch is completed by means of the *genital branch* of the genito-crural trunk, which innervates the cremaster.

In boys the cremaster muscle acts in many cases rhythmically with the other abdominal muscles, so that the testicles rise during inspiration and fall during expiration, as occurs in the ram, the goat, and the dog, in which animals section of the cord arrests this movement.* A like rhythmical activity is sometimes seen in adults, but usually it is lost before the age of twenty. Most young lads and many adults enjoy voluntary power to raise the two testicles at once, and a few persons can at will elevate either testicle singly.

In very few, however, even of the young, is the control of the will equal over both testicles. One rises quicker or more completely than the other, and nearly always one hangs lower than the other at all times.

The cremaster-reflex I described simply in 1872 as capable of being awakened by a touch or pinch of a definite region of the thigh extending from the groin nearly to the knee. Jastrowsky in 1875 describes this reflex as arising on irritations of the inner side of the thigh, and that it is best obtained in the area of the saphena major nerve about a hand's breadth above the internal condyle in the notch between the inner border of the sartorius and the vastus internus. Rosenbach's and Hinze's papers I have been unable to obtain, and do not know how far these authors have studied the limits of this reflex. According to very careful observations made by Drs. Sinkler, Alison, and myself, the region within which we are capable of causing cremaster movement when the skin is touched or pinched is quite extensive in a lad of six to ten years, in whom it is best seen. To learn its limits the observer must bear in mind that certain precautions are necessary. As the scrotum acts at once in a cold atmosphere, and by drawing up the gland obscures the results of cremaster motion, it is well to examine the phenomena when the parts are relaxed and pendant, in a warm room, and this is especially needful in young and strong adults in whom the scrotal purse acts well and steadily.

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* Jastrowsky, *Berlin. klin. Wochenschrift*, Aug., 1875. "On Cremaster-Reflex."

It is also desirable to make the examination so as not to tire the muscle or exhaust the centres, as happens in adults more than in boys. In young lads the cremaster-reflex is easily caused by touching or pinching the whole thigh, with the exception of a band of skin which nearly always may be represented as forming the postero-lateral third of the circumference of the thigh. Within this area no touch or pinch causes cremaster reflex until as we approach the edge of the imaginary boundary, it begins to show and increases as we come around the thigh, until we enter a band of excessive sensitiveness. This region, bounded by the groin and perinæum, is broad above and narrows below, and ceases at or below the knee. Within this area, according to Jastrowsky, lies a limited and most sensitive region. I think him wrong, however, as to this being *always* the most excitable region, since in many people—boys as well as adults—the best cremaster-reflex is had from irritations addressed to parts on the inner aspect of the thigh, and very high up.

These, then, are the usual limits of the cremaster-reflex region, although it is sometimes far less and sometimes far more extensive; but extreme limitations of its area are more apt to occur in adults. When least extensive in the healthy boy it is found to cover the inner third of the thigh, and to cease anteriorly near the middle line of the front of the thigh, and also to fail us above the knee. When largest, it covers the lower third of the belly, all of the space first mentioned by me as its usual territory, and descends down the inside of the leg—over a space which narrows as it goes down, and sometimes includes a large part of the calf of the leg.

In adults, as I have said, the excitor region becomes more restricted. When smallest it occupies a space on the inner thigh, which is broad above and sometimes begins at the line of the perinæum, and sometimes a little below—is irregular in shape, but commonly narrows below and ends above the inner side of the knee. In a few people the excitor region is even less than this, and in numbers of adults it does not exist at all. Jastrowsky thinks that cremaster-reflex is apt to be lost in the fat. I am sure that in old age it is diminished, or even destroyed, and that something in adult life, perhaps the constant contact of our clothes, lessens its activity—as I have never seen it absent in the young, and have many times seen none in vigorous adults.

Wearing of trusses, old hernia, and extreme cases of varicocele, all lessen or destroy it, and as a rule, though not a constant one, when the cremaster-reflex is absent the volitional control is feeble or absent; while on the other hand, a very few healthy people who move the testicles easily by will have no cremaster-reflex.

It is, I think, most remarkable that the excitor region, never, as far as I have seen, includes the scrotum or penis.

I am quite well satisfied that the cremaster-reflex varies from day to day in the same region. I mean that it varies not only as to intensity, but even in some persons as to its existence, and for this there may be some easily conceivable causes. I shall also be able to show that it exhibits like variations in some cases of disease.

The readiness of the reflex response differs also in different persons. In boys the least touch anywhere in the excitor region causes an instantaneous and very complete movement of the testicle, which remains drawn up as long as the irritations are repeated. It is in fact very difficult in the young to wear out the power of reflex response. In the mature adult, however, the cremaster movements are more tardy and less complete, and in some persons there is no movement when we merely touch the skin—a decided pinch of the irritable region being needed to cause motion of the testicle. In the grown man having a good capacity of reflex cremaster motion, when we pinch or touch the skin, at first the testicle rises well, but as we continue to excite the same space, the cremaster responds more and more feebly until at last it entirely ceases to move, no matter how severely the skin be irritated. If now we touch or pinch the skin an inch or two away, the testicle is again drawn up, thus showing that we have worn out the capacity of the skin nerves and not that of the motor centre or of the muscle itself. Returning in a few minutes to the exhausted area we find it again capable of evoking cremaster motion. In others, the first effort to produce the cremaster-reflex totally fails, and it is only after a number of repetitions of the excitation that we succeed in bringing about movement of the testicle.

As a rule which has infrequent exceptions, irritation of one side produces unilateral movement of the testicle of the same side. There are two forms of violation of this law. In the first, irritation of one thigh causes motion of the testicle of the same side and also, a moment later, less complete action on the testicle of the opposite side. In the other case, touching or pinching certain parts of the inner and usually the lower half of the thigh causes reflex-cremaster motion on the other side only; while like irritations in other parts higher up give rise only to unilateral activity on the same side.

The above statements sum up briefly all that I have been able to learn in regard to the normal cremaster-reflex. The extreme irregularity and uncertainty of this reflex in health, prepares us to find that it is of very little value in disease, and so far as I have carried my studies they but serve to illustrate this view. Jastrowsky—op. cit.—says that in hemiplegia there is sometimes no cremaster-reflex on either side during the stupor, but usually, on the day after attack, there is no motion on the hemiplegic side, and increased cremaster-reflex on the sound side—or rarely—irritation of the palsied side gives rise to crossed reflex, which lessens as the palsy improves, but may last for years. If, then, the palsied side becomes hyperæsthetic the reflex relations are apt to be reversed and the lame cremaster acts best.

Hinze (*St. Petersb. Med. Wochenschrift*, No. 35, 1876) quotes Rosenbach (*Arch. f. Psych.*, VI., 845), to the effect that in 10 cases of hemiplegia cremaster-reflex and adominal-reflex were absent on the paralyzed side. The ages are not given. Of his own 21 cases the cremaster-reflex was absent in 18.

My own observations correspond in substance with these. In four cases of recent and very decided hemiplegia, the cremaster-reflex was lost on the palsied side during the first few weeks.

I have met with the following cases of this palsy exhibiting a crossed reflex :

Mr. L., æt. 60, had a right hemiplegia which has so far recovered that he remains slightly lame in arm and leg. The tendon-reflex is very good. The cremaster-reflex excellent on both sides, but a sharp pinch of the left inner side of the thigh causes first a good response from the left cremaster, and about two to three seconds later a slower but complete movement of the right testicle. No other than a limited region of skin gave rise to this phenomenon—which may have been a normal peculiarity.

No. 1, æt. 42, left hemiplegia from embolism; no hemianæsthesia; cremaster-reflex, right, very good; cremaster-reflex, left, good reaction of both cremasters to touch of thigh. When the irritation is frequently repeated the left cremaster ceases to respond and the right still moves well, but a sharp pinch of left thigh again gives rise to bilateral cremaster action. Pinch of left knee inside, and of inner side left calf, causes left cremaster alone to move.

No. 2. Syphilis; albuminuria; right hemiplegia, complete; nearly perfect recovery; no hemianæsthesia; cremaster-reflex, right, good; cremaster-reflex, left, good on upper inner side of thigh for touch and pinch. The same excitations of lower third of inner thigh give rise to bilateral cremaster movement.

No. 3 is a very curious case, and is the only one on record, as yet known to me, of clonic convulsive movements of the cremaster. It will induce me to ask attention to the condition of this muscle in epileptic spasms.

The patient, a man æt. 72, a sea captain, had mitral disease and indurated arteries. I saw him in consultation with Dr. Betts. He was attacked March 17 with left hemiplegia, which, although complete, became rapidly better. March 24, he could move the leg and arm freely. At 4 A. M., he was seized with loss of consciousness, but without any new motor disturbance and without convulsion. When seen by me, at 3 P. M., he was moving all his limbs uneasily. He felt a pinch and showed annoyance in his face. He swallowed well but lay most of the time with closed eyes, the pupils sensitive to light and equal in size.

Cremaster-reflex, right, good, to touch. A pinch caused bilateral cremaster movement tardily on left. At intervals the left cremaster muscle exhibited a singular state of incessant motion. The testicle was drawn up, then fell, then rose and fell rapidly, while the right remained at rest. The cremaster in the belly-wall could be seen to be in almost constant motion, and the lower third of the abdominal wall was also twitching with some slighter display of like movements in the anterior face of the upper fourth of the left thigh. The urine dribbled away from an empty bladder.

PARALYSIS OF INFANCY.

E. S., æt. 3.—Two years' duration. Limited to right leg. There is some slight atrophy of the thigh muscles, but E. S. can move usefully all the leg and thigh muscles, although the triceps has not full power. Tendon-reflex perfect right and left; cremaster-reflex very good over an unusually large area of skin.

H. E., æt. 7.—Four years' duration. Complete loss of power to extend right thigh; with great wasting; can extend and flex foot; left leg sound; tendon-reflex, right, absent; tendon-reflex, left, good; cremaster-reflex, right, good; cremaster-reflex, left, good, and is accompanied with crossed reflex.

J. T., æt. 6.—Paralysis with atrophy of right leg for one year. The thigh muscles move well and are but little wasted; the leg muscles are completely palsied; tendon-reflex was not studied by the observer; cremaster-reflex, right and left, good.

J. M., æt. 6.—Palsy for five years. Left leg only affected; atrophy of thigh and leg muscles (extensors); cannot extend leg or foot at all; muscles of calf partially palsied; plantar reflex, good; tendon-reflex, absent, right and left; cremaster-reflex, absent, right and left.

W. McC., æt. 4.—Palsied two and a half years. General palsy at first and limited to right leg in five weeks; no muscular power in right leg or thigh, save just to stir the toes; leg wasted; tendon-reflex, absent, right and left; cremaster-reflex, absent, right; cremaster-reflex, slight, left.

The cremaster-reflex was studied in four cases of incomplete paraplegia, with more or less spastic contractions of the muscles.

THE ERB-CHARCOT GROUP.

No. 1, æt. 42.—Typical case of the Erb and Charcot symptom-group, with extreme tendon-reflex in many situations; the cremaster-reflex, right, a trace; cremaster-reflex, left, none.

No. 2, æt. 43.—Well marked case. Tendon-reflex exaggerated; cremaster-reflex, right, good—not unusual; cremaster-reflex, left, good, and accompanied by crossed reflex, *i. e.*, left thigh irritation gives bilateral cremaster-reflex.

No. 3, æt. 70.—Typical case of old spastic paraplegia, with convulsive actions in legs on irritation of soles. Exaggerated and violent tendon-reflex; cremaster-reflex, none right or left.

No. 4, æt. 56.—Posterior sclerosis, complicated as it advanced with rigidity of legs; tendon-reflex, exaggerated; cremaster-reflex, right, a trace; cremaster-reflex, left, a trace.

No. 5, a man æt. 60. Much loss of sensation; general wasting of legs; lessening power; feeble bladder; no eye lesions; no neuralgia; staggers with closed eyes; slight rigidity; tendon-reflex exaggerated; cremaster-reflex entirely absent; abdominal-reflex, absent; plantar-reflex, excessive.

The latter case carries us into the ataxias.

There were studied ten cases of well marked posterior sclerosis—none of them in the paralytic stage.

No. 1, æt. 47.—Old case—has lasted 15 years. Tendon-reflex, none; cremaster-reflex, right, none; cremaster-reflex, left, none.

No. 2, æt. 42.—Seven years' standing. Tendon-reflex, none; cremaster-reflex, right, a trace; cremaster-reflex, left, none.

No. 3, æt. 40.—Three years; mild case; slight neuralgia; no eye symptoms, or bladder troubles. Tendon-reflex, right, none; tendon-reflex, left, quite distinct; cremaster-reflex, right, a trace; cremaster-reflex, left, none.

No. 4, æt. 17.—Posterior sclerosis with rare epileptic fits. Tendon-reflex, none; cremaster-reflex, right, none; cremaster-reflex, left, good.

No. 5, æt. 38.—Rapid case, one year's duration. Tendon-reflex, none; cremaster-reflex, right, none; cremaster-reflex, left, a trace.

No. 6, æt. 38.—Posterior sclerosis of three years' duration. Tendon-reflex, none; atrophy of right testicle from mumps, cremaster-reflex, right, good, but varies daily and is easily exhausted; cremaster-reflex, left, none; no left testicle, but well seen in the belly-region of cremaster on tickling thigh.

No. 7, æt. 48.—Six years' duration. Tendon-reflex and cremaster-reflex absent.

No. 8, æt. 42.—Early stage. Tendon-reflex, none; cremaster-reflex, right, good; cremaster-reflex, left, perfect.

No. 9, Mr. N., æt. 35.—Commencing locomotor ataxia. Stands well, eyes shut; tendon-reflex, none; cremaster-reflex, right, is slight; cremaster-reflex, left, none; the left being the worst side.

No. 10, æt. 60.—Posterior sclerosis, earliest stage. Slight neuralgia; no eye symptoms; tendon-reflex absent; cremaster-reflex perfect on both sides.

Except in Cases 8 and 10 the cremaster-reflex was lessened or lost in all of the instances given, but had not the certainty of the tendon-reflex, which was lost in all cases save one, and in that was absent in one leg.*

In a case of dementia paralytica, æt. 30, the tendon-reflex was utterly gone; cremaster-reflex was good, but not perfect.

In a case of acute paralysis of the adult, limited to the right leg and quite complete, the tendon-reflex on right was absent; *good* on left; cremaster-reflex, right, good; cremaster-reflex, left, fair.

It is clear enough, from what I have here said, that the extreme uncertainty of the cremaster-reflex in healthy adults, its variability, as well as its normal irregularities, such as double-reflex, and single crossed reflex, make it of little value in symptomatology—a conclusion re-enforced by all that I have seen of it in disease.

In ataxia it is not lost as is tendon-reflex, nor in spastic spinal paralysis is it increased. In hemiplegias its history is curious rather than useful, and my conclusion is, that cremaster-reflex has as yet little or no value in symptomatology. The frequency of crossed reflex in hemiplegia is very interesting, as well as the fact of the area for double excitation being usually on the left side. Except in extreme cases of infantile palsy the cremaster-reflex does not seem to be disturbed, but in the graver examples of general loss of power in a limb, the cremaster-reflex appears to be absent or weakened on both sides. A larger experience is needed as to this point, since my attention having been but lately given to it I have here noted only five cases.

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* The tendon-reflex has been examined with care at my clinic, and in my private practice in a far larger number of cases, and may, I think, be regarded as one of the most sure, and also one of the earliest signs of uncomplicated posterior sclerosis.

Case 3, of this series, is the only ataxic I have seen who had any remnant of tendon-reflex.

Jastrowsky expresses his surprise that a reflex arc, which is presumed to complete itself in the lumbar cord, should be broken by cerebral disease causing hemiplegia. I had myself made a like reflection before reading the abstract of his paper, and I yet find difficulty in comprehending that, while the plantar-reflex should be exaggerated, the cremaster-reflex should be lessened or lost.

To suppose that it alone of all reflex phenomena should be diminished by removal of cerebral influence seems to me so inconceivable, that I would prefer to believe that the cremaster-reflex track is completed in the brain—a somewhat violent supposition, but without which these anomalous phenomena seem to be incomprehensible.

ART. II.—TEA DRINKERS' DISORDER, OR TOXIC EFFECTS OF TEA.

BY W. J. MORTON, M. D., NEW YORK.

A CASE occurring in practice lately drew my attention to a special train of symptoms associated with the excessive use of tea, meaning by this term the ordinary infusion of tea leaves (*Thea sinensis*), drank daily at our tables.

Around the details of this single case, other data pointing in the same direction, accumulated. One of the most interesting, perhaps, of these was the fact that there exists in New York, as well as in many other parts of the world, a *class* of people which suffers from what would seem to be a specific train of morbid symptoms, occurring in the line of its regular occupation or business (and constituting an "occupation disease" as fairly as does "painters' colic," "glass grinders' disease," or any other of this same nature). The class of people alluded to is that of the "tea brokers," or "tea tasters."

From their number, evidently, was to be obtained the clearest observations as to the toxic or excessive effects of tea. I regret that the limited time at my disposal has not enabled me to make more extended records. But even with time, a more serious drawback to inquiry seemed to exist in the extreme unwillingness of the "tea tasting" fraternity to give their experiences. This arose from the belief that publicity given to any harmful effects which might under any circumstances arise from tea would injure the tea business. I have, however, personally seen five cases, inclusive of one already published in the "Neurological Contributions,"* and these are enough for my present purpose. These cases, and additionally the results of some experiments upon myself, form the basis of this paper, which is presented as a simple contribution to the subject, and in no wise as a compilation from the abundant literature upon tea already existing. But before proceeding further, a few words upon the general subject of "tea tasting."

The pernicious effects of tea tasting upon many of its followers, are well recognized by all of their number. It seems to be accepted among them, without discussion, that many break down and are obliged to give up the business, or else pursue it with much caution and at constant inconvenience to their health. And those of the public who are at all familiar with the facts, entertain the same view. Indeed, I may say that if I were now to express an opinion, based upon my present, it is true, not yet sufficiently extensive information upon the subject, I should feel inclined to say, that no one engages for several years in the profession of "tea tasting" without suffering both immediate and permanent harm to health.

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* "Neurological Contributions." By Prof. Wm. A. Hammond, assisted by W. J. Morton, M. D. New York: Putnam, 1879.

This feeling is well illustrated in the remark of a prominent wealthy tea merchant, who said, "I would rather give a hundred thousand dollars than have my son become a 'tea taster.'"

At first glance, to witness the operation of tasting, it would hardly seem possible that the very small amount of tea used at any one time could result in harm. This amount is only equal in weight to a five cent piece, about fifteen grains. About two ounces of well boiled water are turned on to this, and the infusion is allowed to "draw," perhaps, fifteen minutes. Of this infusion the taster takes but a few spoonfuls into his mouth, and often spits it out again; he also at intervals inhales the steam. But the harm comes, of course, from the constant repetition of these acts.

I have been a little explicit in relating views as to the prevalence of injury to the health of "tea tasters," because of certain grave criticisms to the effect that the symptoms are due to the use of alcoholic stimulants, to dyspepsia, to hypochondria, in short, to anything but tea. But, according to my belief, the list of causes of this nature might be largely added to, and, when complete, be found to range itself in the relation, not of cause, but in that of effect — the effect of tea itself.

The cases which I propose to relate exhibit the extreme physiological action of an infusion of tea. They are cases of acknowledged excess; and in this lies their interest, for by familiarity with the symptoms of excess, we shall be able to thread our way back to those of moderation, and to point out, it may be, that what many perhaps consider moderation is, in reality, abuse, and that certain symptoms put down as "nervousness," "nervous irritability," and "nervous exhaustion," as well as the more clearly defined ailments of dyspepsia, hypochondria and hyperæmia, may sometimes be attributed to the misuse of a common domestic beverage. Indeed, I am forced to think that many people, unconsciously to themselves and to their physicians, suffer from a train of symptoms due to tea (or its congener, coffee). We often find people taking tea to relieve the very set of symptoms which its abuse, as will be shown later, produces; and it is often the fact that patients date their recovery from a dyspepsia or nerve exhaustion from the time when they gave up their tea; though the same, of course, may be as truly said of alcoholic drinks or of tobacco. But owing to the definiteness of the symptoms of alcoholism and nicotinism, and to the baneful effects which their abuse entails upon society, these two substances have been studied by the physiologist and the temperance reformer, and their dangers recognized, even if they are not shunned.

This has been hardly the case with tea. The injuries which it inflicts are so subtle, of such a general nature, and so easily confounded with other causes of harm, that they are unobserved. The very comfort which it showers upon mankind, dims our appreciation of its power for evil; for in the midst of the many who are benefited by it, those who are injured escape notice. The cases which follow, combined with experiments upon myself, will, however, bring out into the clear light symptoms due alone, I think, to tea; and it is no argument against its causative force as a factor in producing these symptoms, to say that alcohol, or tobacco, or something else, may produce almost identical ones. This, indeed, holds true for any one individual symptom, be it vertigo, mental irritability and exhaustion, dyspepsia or some other; but it does not hold true for the group. It is to the group alone to which I wish to call attention. Medicine has coined such words as Alcoholism, Plumbism, Iodism, and many of this class, in order to express its recognition of the toxic effect of these agents by a concise and special term. Is there not equally a well defined set of symptoms which represent the full and toxic effects of tea?

I have ventured, at least, to use the expression, tea drinkers' disorder in this sense, and I think that what follows will sustain this view.

Case I. A. B., a young gentleman aged 30, somewhat sallow and thin in flesh, but apparently in general good health. He had been a "tea taster" as well as broker for about eight years. He estimated that about a half a pound of tea, weighed before being made into infusion, enters the mouth in a day.

I have arranged his symptoms under the headings of immediate and chronic.

The immediate effects upon him are as follows: In about ten minutes the face becomes flushed, the whole body feels warm or heated, and a sort of intellectual intoxication comes on, much the same in character, it would seem, as that which occurs in the rarefied air of a mountain. He feels elated, exhilarated, troubles and cares vanish, everything seems bright and cheerful, his body feels light and elastic, his mind clear, his ideas abundant, vivid, and flowing fluently into words. He has found from experience that the workings of his intellect are really more clear and vigorous than at any other time. This is not a delusion on his part, for at this time he can "talk a man over" and make a more advantageous bargain than at others.

At the end of about an hour's tasting a slight reaction begins to set in; some headache comes on; the face feels wrinkled and shriveled, particularly about the eyes, which also get dark under the lids.

At the end of two hours this reaction has become fully established, the flushed, warm feeling has passed off, the hands and feet are cold, a nervous tremor comes on accompanied with great mental depression. And he is now so excitable that every noise startles him; he is in a state of complete unrest and mental exhaustion; he has no courage to do anything; he can neither walk nor sit down owing to his mental condition, and he settles into a complete gloom. His body in the meanwhile does not feel weary. Copious and frequent urination is always present, as also certain dyspeptic symptoms, such as eructations of wind, sour taste, and others.

The above described immediate effects follow a single afternoon's tea tasting. They may be summed up briefly, as, excited circulation, intellectual intoxication, with actual increased vigor of mind power, increased urination; then a period of collapse indicated by cold extremities, tremulousness, mental irritability and anxiety. It will be several days before this condition of affairs is amended. And at this time the temptation to take alcoholic stimulants is very strong.

The chronic effects are few and decided. Headache is frequent, principally frontal and vertical; a ringing and buzzing in the ears is very constant; black spots often flit before the eyes, and he sees flashes of light. Vertigo also is very persistently present; he cannot look up at a clock on a steeple without staggering. Insomnia exists to a considerable extent; he seldom has a good night's sleep and he dreams much, but his dreams are of a pleasant character; he sometimes sees visions when not sleeping. Dyspepsia is more troublesome than any of the foregoing three symptoms. This the patient assigns strictly to tea tasting, since it is made worse by tea, and improves when he abstains from it, though now becoming confirmed. His appetite is captious, he feels heavy at the epigastrium, he has eructations and a sour taste and finds that certain kinds of food distress him. He has a frequent gurgling, and is in the habit of "working" his whole chest and abdomen to make the gas pass on.

His mental condition is peculiar. He lives in a state of dread that some accident may happen to him; in the omnibus fears a collision; crossing the street fears that he will be crushed by passing teams; walking on the sidewalks fears that a sign may fall, or watches the eaves of the houses, thinking that a brick may fall down and kill him; under the apprehension that every dog he meets is going to bite the calves of his legs; he carries an umbrella in all weathers as a defense against such an attack. He often dreads entering his office for fear of being told that some business friend has failed; and in short, lives in a state of constant foreboding of some impending evil. At times his left leg drags and feels numb, and he is conscious of an unsteady gait. He has also often a twitching of the muscles of the face and eyelids.

The chronic effects as above described as distinguished from the immediate effects are in brief, vertigo, headache, insomnia, dyspepsia, mental depression almost amounting to delusions, and also some slight subjective and objective signs of a central disturbance of both sensibility and motility.

I omit negative evidence indicating that otherwise than as related, he is in sound health.

A certain group of his symptoms point to hyperæmia of the brain, another to disorder of the digestive function, and still another to morbid alterations of intellection, sensibility, and motility; all taken together presenting, as I venture to suggest. a clinical picture of tea poisoning.

Case II. Mr. P.—Has been in the tea business about five years, and frequently "tastes" tea, though always now with reluctance, since he is convinced that the practice is doing him harm. He is a strong looking man of twenty-five, of the sanguine temperament, and does not use alcoholic stimulus in any form. Convinced in his own experience of the detrimental effects of tea tasting upon himself, he is endeavoring to judge of teas he wishes to buy, by smelling of the steam arising from the fresh infusion; but even this practice produces many of the disagreeable symptoms which he wishes to avoid.

The *immediate* effects upon Mr. P. of tasting are as follows:

Within a few minutes an urgent desire to urinate. That this is not occasioned by the hot water he is positive from the fact that in all cases it depends upon the variety of tea tasted. With certain teas there is no such desire—with others, particularly Japan tea, it ensues immediately. Other tea tasters corroborate the existence of this peculiarity in Japan tea, and if further proof were needed the experience of a firm which employed nearly a hundred girls in putting up spices, might be cited. On certain occasions the superintendent has served out Japan tea to his employees, but was obliged to abandon its use because they lost so much time in relieving themselves from the results of the extreme diuresis produced.

The stimulating or intoxicating effects quickly ensue; with a feeling of bodily warmth and freshness comes on a period of intellectual clearness; he feels in an improved condition of both mind and body, and if he can stop tasting at this point and take no more tea he suffers but a slight reaction. But even at this early stage a slight tremulousness has appeared in all parts of his body, particularly in his hands, which tremble continuously when he attempts to take hold of anything. This is a tremor exactly like that of the habitual drunkard, but in Mr. P.'s case it is certainly only associated with the drinking of too much tea. Ordinarily, as I can affirm, his hands are perfectly steady, and, as I have said, he never uses alcohol. Accompanying the tremor are certain paræsthetic symptoms of formication, numbness, and feelings of heat and cold, and also slight muscular twitchings around the eyes, nose, and cheeks.

If he has been obliged to taste a considerable amount of tea, a very uncomfortable reaction now sets in. His face has already begun to feel shrivelled and wrinkled about the eyes and cheekbones, and now the further very distressing symptom of headache, chiefly frontal, appears, as also some slight nausea. An extreme sense of weariness has taken the place of his exhilaration, and he is conscious of great irritability of temper; his face is now very pale, and the "drawn" feeling about it still persists.

At the end of several hours, that is to say, after an excess of tasting, this sense of weariness has become depressing—it is complete languor; as he describes it, "an absence of hilarity—disinclination to make a joke or enjoy one." In Case I., a patient of a more hypochondriacal temperament, it is curious to note that this stage was one of melancholy and gloom. Mr. P. says, that tea tasting "makes a very peculiar draft on the strength," and that his family always know when he has been making a purchase of tea, because he comes home pale, tired and utterly exhausted. He also agrees that at this period there is a strong craving for alcoholic stimulants, and that it is useless to take more tea, as that only makes matters worse. After a night of sleeplessness and a subsequent good night's rest, he has quite recovered from the above described train of immediate effects of tea tasting. In his case the chronic effects appear to be certain symptoms of dyspepsia, which he refers to as a "gone feeling" at the epigastrium, and as a "horrible craving for food." He has also other characteristic symptoms, pointing in the same direction. These, as in Case I., improve upon ceasing tea tasting, and are aggravated by it; but I shall take occasion later to point out that I do not consider dyspepsia in the light of a cause of the symptoms seen in tea drinkers' ailment, but simply as one of many effects produced by tea. Mr. P. has suffered from no marked mental symptoms.

Case III. Mr. D., thirty years old, and a very strong looking man. He has been "tasting tea" for several years. The following facts were brought out in conversation. From his experience, Mr. D. thinks tea is "very injurious stuff." He is endeavoring to go by the smell now, to see if he cannot avoid the discomfort tasting occasions him. It seems to affect his stomach chiefly; when he first begins on a new lot of samples, he is always nauseated, and frequently vomits. He feels a brief period of exhilaration, and a very intense reaction; he gets so utterly depressed, that he is obliged to take brandy to keep up his strength. (The other two cases, it will be remembered, did not use any alcoholic stimulants.) He drinks in moderation daily.

Dyspepsia was unknown to him before he began tea tasting, now he is a chronic sufferer. After each tasting, he has, to use his words, "an all-gone feeling," and a craving for food. He thinks it creates an artificial hunger. He never sleeps through all of the night—always feels wakeful, watchful and apprehensive of danger or trouble; he starts at the slightest unexpected noise; he suffers much from tremulousness. He does not use coffee, or smoke.

In reading over such a case, one may well ask if this is not alcoholism. Of course it is hard to decide. But since there is a known abuse in tea tasting, since others who do not use alcohol (Cases I. and II.) have the above symptoms, and since, as I shall relate later, I have produced them in myself, in a series of experiments undertaken to prove the same toxic effects, I am positive that they are due to tea alone.

Case IV. Mr. W. Of this case I could only gather a few points. They were briefly these: After each "tasting," he is certain to have, what he calls, a sick headache, and also a sleepless night. He suffers much from dyspepsia. When he first takes tea, it flies to his head and eyes, his head "swims," bright lights dance before his eyes, the lids feel heavy, and he soon settles into a steady headache of at least twelve hours duration.

Mr. W. was obliged at last to give up tea tasting as a profession, though he now uses, and is very fond of, his daily cup of tea.

Case V. Mr. D. H. I will not relate the details of this case, which presents essentially the same features as the preceding ones, with this additional peculiarity only, that Mr. H. is the victim of both panphobia and illusions. He is always what he calls "nervous;" he is watchful of his best friends even; he always tries to keep with people, because of a horror of solitude, and yet he lives in constant fear of their doing him some bodily harm. If any one walks up quickly behind him, even in frequented Broadway, he looks around in fear, and often steps aside to let them pass. After close questioning, I felt certain that he suffers rather from illusions than hallucinations. He frequently thinks he sees a mouse running about on the floor; but this he generally finds out to be a shadow or some stationary object which his eye has caught in turning suddenly away from it. He often ducks his head to avoid the sparrows in the street, thinking they are missiles, either falling or thrown by boys.

Mr. H. used to drink wine and lager beer, but never spirits. Under medical advice, he has confined himself to a pint of claret daily, now for six months, so that alcohol cannot in the least be considered in the light of a cause of his symptoms. There was but one advice to give him, and that was to give up his business of tea tasting immediately, or make up his mind to become a lunatic.

EXPERIMENTAL INVESTIGATIONS.

With the view of clearing up and verifying some points which arose in this inquiry, and of ascertaining in an approximate manner, what the effects of tea drinking in excess, upon the human economy were, I resolved to institute some experiments upon myself.

I took from four to eight drachms of gunpowder or green tea, and had it made into an infusion with a pint of water. This I drank at intervals during the day, and continued it steadily for seven days, not altering otherwise my ordinary habits of life, which are temperate and abstemious. My mental work was about the same as usual; it consisted for the week in light medical duties, translation from German, and writing this paper. My chief points of inquiry were:

1. The effect upon the nervous system.
2. The effect upon the waste of tissue, as shown by the urine, which was subjected to a daily quantitative examination.

I give a brief daily record of the week, during which I subjected myself to tea. The mean temperature of weather was 69.7° F.

Wednesday, June 11*th*, 1879.—Dry, hot day; pulse 74; resp. 18; temp. 99.

In forenoon took ʒ iv. of green tea in infusion. Pulse at noon 96; resp. 26; temp. 100.

Took ʒ iii. at 4 o'clock.

At 5 o'clock, pulse 100; resp. 27; temp. 100. Nausea; severe pain in both temples; no exhilaration, but rather dullness of brain.

At 6½ o'clock, severe headache, with throbbing. Pulse 84; resp. 26. Severe headache continued until 1 o'clock that night.

Anal. of urine.—Sp. Gr. 1018; reaction, acid; quant. 40.57; urea 591; ur. ac. 5.065; SO_3, 21.62; P_2O_3, 53.25; Cl. 65.5; Na. Cl. 108.

Thursday, June 12*th*.—Hot day. Slept 7 hours. Pulse, when half awake, 80; resp. 17; temp. 99½, which fell one degree after a cold-water bath. Head felt sore from yesterday's headache.

Took ʒ iii. tea at 9 o'clock. No ill effect; mental work till noon. At 2 o'clock took ʒ iii., with sugar and ice. At 3 o'clock felt delightfully well, with contentment and placidity of mind; legs felt light and strong, and back felt firm on the thighs. The feeling of lightness, comfort and strength combined brought to mind often the comparison to a man made of the metal aluminium. Strong desire to walk, and though others complained of heat, I felt cool, and walked two miles rapidly, and was then unwilling to stop. The body moved so lightly, as to give almost the sense of floating. No confusion of intellect, but rather much clearness of insight; whatever knowledge was in the mind, seemed very available, as I noticed in examining patients at the afternoon clinic. Contrast to yesterday certainly was very marked. At 7 o'clock began to feel a sense of tightness, like a band around the head. Took ʒ ii. of tea, and at 8 o'clock, ʒ iii.

At 9 o'clock severe pain in temples, and no further exhilaration.

At 12 o'clock, pulse 84; temp. 99¼; resp. 25. To bed.

Qual. and quant. anal. of urine.—Sp. gr. 1024; react. quite acid; quant. 36.352; urea 496.5; uric ac. 8.94; SO_3, 37; P_2O_3, 62; Cl. 88.15; Na. Cl. 145.25.

Friday, June 13*th*.—Morning. Pulse 76; resp. 18; temp. 98$\frac{3}{4}$. Took ʒ viii. of tea during the day. Evening. Pulse 84; resp. 21; temp. 99. During all this day felt restless and "nervous." I noticed now, for the first time, a general tremulousness in hands, arms and legs, also a tired feeling, and twitching of the facial muscles, had also a feeling of dread, *i. e.*, a fear of impending evil. Slept very poorly.

Anal. of urine.—Sp. gr. 1024, very acid; quant. 33.14; urea 498; uric ac. 10.93; SO_3, 32.2; P_2O_3, 78.75; Cl. 75; Na. Cl. 123.5.

Saturday, June 14*th*, 1879.—Morning. Pulse 84; resp. 18; temp. 99$\frac{3}{4}$. Slept 7 hours the last night. Took ʒ iv. of tea at breakfast. Soon felt very heavy in the upper eyelids, and some vertigo and dimness of vision.

At 12 o'clock, body elastic and felt contented in mind, but found that I was very nervous. Several friends noticed this, a condition which I had never before experienced. Every sudden noise made me start; the whistle of a speaking tube, the falling of a board, or a sudden remark. But with the feeling of "nervousness," there was no feeling of irritability of temper; on the contrary, placidity and a delightful intellectual clearness, ideas came rapidly, and I found myself wishing that such a condition would continue. Body felt cool and fresh; comfort, exhilaration and wakefulness seemed to be the condition of affairs.

4 P. M.—ʒ ii. Eyelids immediately felt stiff and heavy; later, some nausea, perspiration, tremulousness, and headache; fingers felt numbed, as if wrapped in cotton-wool; felt very heavy about the eyes, also hot, flushed and uncomfortable; ears rung, buzzed and felt full.

Two hours later, felt very miserably, tired, weary, exhausted, trembling and unstrung; then got painfully sleepy, and slept uneasily for 7 hours.

June 14. Analysis of urine.—Sp. gr. 1022, very acid; quant. 30.94; urea 443.83; uric ac. 5.636; SO_3 21.98; P_2O_5 73.5; Cl. 65; Na. Cl. 107.

Sunday, June 15th.—Pulse 84 all day; resp. 20.

Took ʒ iv. during the forenoon, in two ʒ ii. doses. Mind dull and have a headache; trembling feeling all over; feel weak and trembling at pit of stomach.

After second dose, had profuse perspiration; got into such a nervous state that I had to give up work. The flies seemed to come at me as big as cannon balls; the scratching of my pen on the paper was very distressing, and also the noises in the streets; got very black under the eyes. Concluded not to take any more tea to-day.

Only ʒ iv. taken in all.

Analysis of urine.—Sp. gr. 1026½; react. very acid; quant. 27.90; urea 439.51; uric acid 13.49; SO_3 28.97; P_2O_3 73.69; Cl. 65.43; Na. Cl. 107.74.

Monday, June 16th.—Slept 7 hours, and fairly well. Pulse 84; resp. 18; temp. 98½. Quite recovered from effects of yesterday's tea.

Morning, took ʒ ii. No discomfort.

Noon, took ʒ iii. Made me feel very well.

Evening, took ʒ iii. of oolong tea.

Evening, pulse 90; resp. 24; temp. 100.

Tuesday, June 17th.—Broken sleep of 7 hours last night. M. pulse 84; temp. 99; resp. 24.

My sample of green tea was done, and I, therefore, used strong oolong, or English breakfast tea, which did not seem to have much effect upon me. Walked a mile, and did some mental work. The pleasant effects of the tea were predominant.

Evening.—Pulse 80; resp. 18; temp. 98½.

Quant. analysis of urine.—Sp. gr. 10.20; reaction acid; quant. 32.135; urea 422.88; uric acid 8.408; SO_3 28.77; P_2O_5 63.64; Cl. 59.72; Na. Cl. 98.42

RESUMÉ.

IMMEDIATE EFFECTS OF MODERATE DOSE.

(*a*) Elevation of pulse.

(*b*) Increased respiration.

(*c*) Agreeable exhilaration of mind and body, combined with a feeling of contentment and placidity, and a cool and fresh feeling of the body. *

(*d*) Increased mental and physical vigor.

(*e*) No noticeable reaction.

IMMEDIATE EFFECTS OF EXCESSIVE DOSE.

(*a*) Rapid elevation of pulse.

(*b*) Marked increase of respiration (increased by $\frac{1}{3}$).

(*c*) Increase of temperature.

(*d*) No period of exhilaration, but immediate and severe headache, vertigo, dimness of vision, possibly nausea, heat and flushing of body, ringing in ears, heaviness around the eyes.

(*d*) Mental dullness and confusion of ideas.

(*e*) Severe reaction; exhaustion of mind and body; tremulousness; "nervousness," apprehension of harm and impending evil; sleeplessness; cold perspiration; disinclination to mental and physical exertion. This reaction cannot be relieved by taking more tea.

(*f*) No irritability of temper, in spite of the above disagreeable symptoms.*

CONTINUED EFFECTS OF EXCESSIVE DOSE.

Additional to the *immediate* effects of both the small and large doses, just narrated—and deduced from the five cases reported and effects upon myself:

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* Irritability of temper like dyspepsia belongs in the category of symptoms produced by long continued tea drinking.

There are "*tea sots*" in every great charitable institution—particularly those for the maintenance of the aged. Their symptoms are, generally, mental irritability, muscular tremors and sleeplessness. Interesting facts in this relation have been brought forward in the most valuable and comprehensive Report of Dr. Henry I. Bowditch, of Boston, to the Massachusetts State Board of Health, upon the "Use and Abuse of Intoxicating Drinks throughout the Globe."

A recent writer, Dr. Bock, of Leipsic, takes very strong grounds in regard to the mental influence of continued tea drinking. We quote merely a sentence or two—"The nervousness and peevishness of our times," he says, "are chiefly attributable to tea and coffee; the digestive organs of confirmed coffee-drinkers"—he elsewhere includes tea in the same rank—"are in a state of chronic derangement which reacts on the brain, producing fretful and lachrymose moods." And again, "The snappish, petulant humor of the Chinese can certainly be ascribed to that immoderate fondness for tea."

(*a*) A continual tremulousness, and extreme susceptibility to the influence of external impressions, particularly noises, together with a feeling of being "unstrung" or nervous.

(*b*) Constipation.

(*c*) Diminution of the amount of urine.

(*d*) A marked influence upon the retrograde metamorphosis or "waste of tissue"—as evidenced in the large and daily diminution in the amount of urea excreted.

I give here in tabulated form the results of the quantitative examination of the urine for the week, with a few qualitative remarks.

The daily analyses were made by my friend, E. Waller, Ph. D., in charge of the Chemical Laboratory at the School of Mines, Columbia College. The urea was measured by Liebig's method with mercuric nitrate: the uric acid by the usual process with hydrochloric acid; the chlorine by a standard solution of nitrate of silver ($\frac{1}{10}$ normal); the phosphoric acid by a standardized solution of ferric chloride; the sulphuric acid gravimetrically by barium chloride in acidified urine.

AMOUNTS IN FLUID OUNCES AND GRAINS.

Mean temperature of the weather 69.7 F. Barometer, 29.83. Humidity, 76. Rainfall, .07 inches.

Date.	Sp. Gr.	Reaction.	Color.	Quantity	Urea.	Uric Acid.	Sulphuric Acid.	Phosphoric Acid.	Chlorine.	Chloride of Sodium.
June 11	1018	acid	high	40.5792	591.36	5.065	21. 62	53. 22	65. 60	108. 11
" 12	1024	quite acid	"	36.352	496.65	8.939	37. 08	62. 08	88. 15	145. 22
" 13	1024	very acid	"	33.140	498.04	10.934	33. 20	78. 78	75. 00	123. 60
" 14	1022	"	"	30.940	443.83	5.636	21. 98	73. 55	64. 96	107. 09
" 15	1026½	"	"	27.900	439.51	13.490	28. 97	73. 69	65. 43	107. 74
" 16	1026	"	"	27.560	445.52	11.798	32. 63	69. 16	62. 37	102. 79
" 17	1020	quite acid	"	32.135	422.88	8.408	25. 90	63. 64	59. 72	98. 42
Average	1022.8	Acid	High	32.67	476.81	7.752	28. 77	68. 16	71. 60	113. 29

The same table calculated by the metric system:

Date.	Quantity.	Urea.	Uric Acid.	Sulphuric Acid.	Phosphoric Acid.	Chlorine.	Chloride of Sodium.
June 11	1200	38. 40	0. 264	1. 404	3. 456	4. 260	7. 02
" 12	1075	32. 25	0. 580	2. 408	4. 031	5. 724	9. 43
" 13	980	32. 34	0. 710	2. 156	5. 116	4. 870	8. 026
" 14	915	28. 82	0. 366	1. 427	4. 776	4. 218	6. 954
" 15	825	28. 54	0. 876	1. 881	4. 785	4. 249	6. 996
" 16	815	28. 93	0. 766	2. 119	4. 491	4. 050	6. 675
" 17	950	27. 45	0. 546	1. 682	4. 132	3. 880	6. 391
Average	965	30. 96	0. 587	1. 868	4. 391	4. 464	7. 356

A glance at the table shows,

1. A decided increase in the sulphuric and phosphoric acid, while the chloride of sodium remains about stationary.

2. A steady daily decline in the amount of urine excreted. This amounted to an average daily decline of about two fluid ounces, or to a total decline for the week of thirteen fluid ounces.

3. A large daily decline in the amount of urea excreted. This amounted to an average daily decline of 24 grains or to a total decline of 168 grains. The most marked decline was that on the first day of 95 grains. This it will be remembered was the twenty-four hours during which I suffered, for twelve hours, from the extreme toxic effects of tea, indicated by severe headache and other symptoms recorded.

These results as regards urea show a much greater diminution in the same time, than do those obtained by Dr. Hammond, and recorded by him in a table similar to the above, printed in the *American Journal of the Medical Sciences*, for January, 1855. In his table the only constituent altered was the urea, which had diminished from 621 to 606 grains or 15 grains in the week. The decline in the total amount of urine excreted was only four ounces. But in my own case far greater amounts of tea were used.

The experiments of Böcker, Lehman, and others, have previously established the fact of the diminution of urea caused by the ingestion of tea, and it is considered established that its use retards the "waste" of the body, as evidenced in the secretion of the kidneys, and thereby diminishes the demand for food.

The results of the table above given agree with the results of other observers, though they seem to be more decided, and this I can only account for by the strength of the sample of tea I made use of.

GENERAL CONCLUSIONS.

1. With tea, as with any potent drug, there is a proper and improper dose.

2. In moderation, tea is a mental and bodily stimulant of a most agreeable nature, followed by no harmful reaction. It produces contentment of mind, allays hunger and bodily

weariness, and increases the incentive and the capacity for work.

3. Taken immoderately, it leads to a very serious group of symptoms, such as headache, vertigo, heat and flushings of body, ringing in the ears, mental dullness and confusion, tremulousness, "nervousness," sleeplessness, apprehension of evil, exhaustion of mind and body, with disinclination to mental and physical exertion, increased and irregular action of the heart, increased respiration.

Each of the above symptoms is produced by tea taken in immoderate quantities, irrespective of dyspepsia or hypochondria, or hyperæmia. The prolonged use of tea produces, additionally, symptoms of these three latter diseases. In short, in immoderate doses, tea has a most injurious effect upon the nervous system.

4. Immoderate tea drinking, continued for a considerable time, with great certainty produces dyspepsia.

5. The immediate mental symptoms produced by tea are not to be attributed to dyspepsia.

In the above experiment upon myself, the whole group of symptoms was produced, with no sign of digestive trouble superadded.

6. Tea retards the "waste," or retrograde metamorphosis of tissue, and thereby diminishes the demand for food.

It also diminishes the amount of urine secreted.

7. Many of the symptoms of immoderate tea drinking are such as may occur without suspicion of tea being their cause; and we find many people taking tea to relieve the very symptoms which its abuse is producing.

ART. III.—MASTURBATION AS A CAUSE OF INSANITY.

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THE frequency with which masturbation is practiced by the insane, and the meagre descriptions of the subject in works on insanity, shall be my excuse for recording a few facts in connection with masturbatic insanity, as they have presented themselves during the past three years in the wards of the Cook County Hospital for the Insane. I shall endeavor to show that masturbation is an exciting cause of insanity, also the relations they bear to each other, and enumerate some peculiar physical conditions associated with the disease.

Is masturbation alone ever the exciting cause of insanity? Although some medical writers go to the extreme of disregarding *in toto* masturbation as an exciting cause, the numerous cases reported of masturbation terminating in insanity in persons of sound mental and physical health, in whom there did not exist the slightest hereditary predisposition, alone would justify us in regarding it as a predisposing, as well as exciting, cause. Prof. Maudsley writes: "And there is, certainly, a characteristic variety of insanity, caused by self-abuse, which makes the patient very like a eunuch in character."

Hence, the importance of early treatment before there are any marked mental complications, and "while motives besides fear are all-powerful."*

The total number of admissions to this hospital, since Jan., 1860, is nearly fifteen hundred—eight hundred males and seven hundred females. Of the males admitted, the exciting cause of insanity was stated as masturbation in forty-nine cases. I regard this number as an exaggeration, the error usually arising from the want of proper testimony at the trials of patients, and a failure on the part of the jury to discriminate between masturbation as a cause, or a mere concomitant of insanity. Altogether too much importance is usually attached to the testimony that the patient does or has masturbated. After making due allowance for all sources of error, there still remains a considerable percentage of cases whose insanity must be regarded as a direct result of masturbation.

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*Dr. Luther Bell, Annual Report, 1844.

The following tables will show the nativities, occupations and ages of all the patients admitted and entered as "Masturbatic Insanity," since the opening of the hospital:

NATIVITIES.		OCCUPATIONS.		AGES.	
America	15				
Austria	1				
Bohemia	1	Laborers	19	Under 20 years	10
England	1				
France	1	Mechanics	14	Between 20 and 30 years.	25
Germany	13				
Ireland	9	Clerks	10	" 30 " 40 "	12
Norway	1				
Poland	1	Not specified	6	" 40 " 50 "	2
Sweden	6				
Total,	49		49		49

The above tables show the ages of the patients, when owing to an impaired mental condition, it became necessary to remove them to an asylum, and does not indicate the commencement of the vice, which antedates the ages given by many years, dating as far back as early manhood, or even the age of puberty.

Thirty-five of the patients admitted were under thirty years of age, and, with one exception, they were all single men. The exceptional case, Mr. H., was a widower; wife died several years prior to his admission. He was father to several children, and perfectly well until after the death of his wife, when he was several times detected in the act. His general health became so impaired, that he was unable to attend to his business, and was removed to this hospital, where he died completely demented, and masturbating very frequently.

The histories obtained usually are similar in many respects. The following will answer as an example, at the same time illustrating the fact that masturbation is an exciting cause of insanity in persons of sound mental and physical health: Mr. A., aged twenty-three years; American; single; eight brothers and sisters, also both parents living and enjoying good health. I succeeded in getting an unusually complete history of both branches of his family, and could not learn of a single case of insanity, epilepsy, or other serious nervous disease. He enjoyed perfect health until fifteen years of age, when he left school to clerk in a store, where he was first noticed to act peculiarly. He proved a source of great annoyance to his employers and associates; his periods of excitement were well marked, and he avoided all society, especially females, when it was possible to do so. As yet, his intellect seemed unimpaired, beyond a slight loss of memory. The loss of memory was for some time the only symptom of mental weakening noticed, and became so marked that it was unsafe to intrust him with any important business.

Being aware of the injurious results following masturbation, he would frequently resolve to discontinue the practice, but his volitional powers became so weakened by his repeated failures at reform, that he attempted to commit suicide, to put an end to his mental suffering. As his general health failed, his nervous system especially seemed to suffer, as was shown by his excitability, twitching of the facial muscles under excitement, and irregular action of the heart, while imperfect textural changes throughout the entire body showed the impairment of the general health. His frequent failures at reform so weakened his will powers, that he was unable to make any further efforts to discontinue the degrading vice. From this time on his general health failed rapidly, the intellect became greatly impaired, alternate periods of excitement and dejection harassed his mind until he died in a state of complete dementia. As is frequently the case, his delusions were self-accusatory in character.

I have seen but a single well marked case of exaggerated conceit mentioned as an important feature in some cases of masturbation. The patient was admitted several months ago with the following history: Mr. B., American, aged 21 years, single, and apparently well, until two and a half years ago; was very bright in school, generally stood at the head of his class, and nothing in his speech or conduct ever led any one to regard him otherwise than perfectly well. Both parents are living, enjoying good health, and there is no history of insanity in either of their families. At the age of seventeen he secured a position as cashier in a large dry goods house, where he worked faithfully, until compelled to resign, owing to a failure of his health. His parents, for the first time, noticed a change in the affections and deportment of their son. He was irritable and subject to paroxysms of ungovernable rage; upon several occasions attempted to do his mother bodily injury. His general health failed rapidly, and he took such dislikes to his friends, and especially his parents, that they were compelled to remove him to a place of safety.

Upon examination after admission, found him poorly nourished, heart and lung sounds normal, heart's action accelerated, tongue tremulous, pupils moderately dilated, marked neurasthenia and loss of memory, especially of recent occurrences. The accelerated heart's action probably is entirely reflex in character, and due to vaso-motor excitability. The cutaneous sensibility, as tested with the æsthesiometer, was found nearly normal. Reflex excitability is somewhat exaggerated, especially marked in the tendo-reflex phenomenon. Tickling the soles of the feet while asleep, caused violent contractions of the leg on the same side, and gradual flexion of the opposite leg. The prepuce was found elongated, and a partial condition of phymosis was found present, but has since been remedied by an operation. He confesses that he masturbated frequently, since fourteen years of age, but is unwilling to make any efforts at reform. In this respect the case is somewhat peculiar, as most of these unfortunates are very anxious to abandon the vice, but unable even to make an effort, for reasons already stated. He is very effeminate in all his habits; constantly carries an open and shut fan, delights in ladies' small talk, spending most of his time in needlework, arranging his toilet for the weekly dances, and other equally effeminate employments.

The corrupted sexual feelings, such as forming morbid attachments for persons of his sex, are quite marked, and it became necessary to remove several patients to different wards to separate him from the object of his regards. The most noticeable feature in the case, and one that attracts the attention of all with whom he comes in contact, is the vain opinion he entertains of himself. The attendants and even visitors have noticed and commented upon it. He flatters himself that the female attendants are enamored with him, and takes great pleasure in enumerating the compliments bestowed on him by the ladies, especially such as refer to his personal appearance and attainments; boasts of his clothing, style of dancing, and is never so happy as when complimented by any one upon a real or imaginary acquisition. His styles of dressing are very eccentric. On his fingers he wears two large rings; one brass with glass setting, the other, several rows of ordinary white beads; and when asked concerning their significance, replied, "I wear them because they are becoming to my peculiar style of beauty." Although very emotional and readily excited to anger and violent demonstrations, he is as readily subdued by the least show of firmness. He carefully avoids obscene and boisterous language on the ward, but is apt to be very familiar with the ladies whenever an opportunity presents. When at home, he attempted to get in bed with a female servant against her will, and when asked for an explanation of his strange conduct, excused himself by saying, that he had been frightened by the lightning accompanying a thunder-storm, and sought protection in the servant's bed.

The prognosis I regard as unfavorable, owing to the impairment of his general health, loss of memory, mental lassitude, weakened powers of attention, neurasthenia, his defence of the vice and approaching dementia. The case probably will terminate in complete dementia and death, unless he succumbs to some intercurrent affection, as pleuritis, pneumonitis or peritonitis, which, owing to diminished vital and recuperative powers, would be likely to prove fatal.

Cases of masturbation terminating in insanity pass through two stages. The first or conscious stage, commencing with the vice, and terminating in the second stage; the second or unconscious stage, is a continuation of the first, when owing to impaired mental and especially weakened volitional powers, the patient is unable to make any further successful efforts at reform. This stage usually terminates in dementia and death, unless some successful means are employed to improve the general health and strengthen the weakened will-powers.

The predisposing causes of masturbation are very numerous; a few of the most important may be enumerated as follows: A nervous temperament; stimulating diet; improper associations and training; obscene literature; an arrested cerebral development (imbeciles frequently are confirmed masturbators); partial phymosis, with hyperæsthesia of the glans penis, where every attempt to uncover the glans is accompanied by an erection, are a few of the most frequent causes of masturbation. How any cause calculated to unduly stimulate or to direct the attention of an excitable youth to the genitalia acts as a predisposing cause, is very apparent.

The general health of insane masturbators is always impaired. They are usually anæmic, with slow or irregular pulse, and all the evidences of imperfect nutrition are present. As the acute symptoms subside, there is occasionally an apparent improvement of the general health, as is shown by an increased weight. This improvement is not real, however, as may be demonstrated by enforcing active bodily exercise, which is always followed by undue fatigue.

The second stage is frequently ushered in by acute maniacal symptoms, with impaired appetite and loss of sleep; delusions of a depressing character are usually present, and homicidal or suicidal tendencies are occasionally manifested.

In a small percentage of cases the intellectual faculties are but slightly impaired, even after a complete loss of memory.

To determine what physical changes the abused organs undergo, I examined the genitalia of twenty-six insane masturbators, and found the following conditions present: Penis and scrotum, so far as external appearances go, normal in eighteen; penis enlarged in five, and atrophied in three of the patients examined. J. G. has a typical masturbator's penis, enormously enlarged and never perfectly erected while experiencing the orgasm with emissions. The penis is rarely well erected in old cases, and is only semi-erected during the excitement accompanying the act. I examined twenty-five of the patients to determine the frequency of stricture of the urethra, passing an ordinary size silver catheter. The catheter passed into the bladder without meeting with any obstructions in nineteen cases; in four it met with a constriction, through which it was passed with little trouble. In two instances the instrument could not be passed without using undue force. As there is no history of gonorrhœa in any of the cases examined, it is safe to conclude that a majority, if not all the strictures, are in some way the result of masturbation.

The slight constrictions I regard as due entirely to a thickening of the mucous membrane lining the urethra; an undoubted muscular element was present in two instances. The hypertrophy is due to a low grade of inflammation commencing in the mucous membrane, and may involve deeper tissues, and the inflammation is probably the result of the unnatural irritation of the parts. The seat of the stricture usually was found about the commencement of the membranous portion of the urethra. The mucous membrane was very vascular, and considerable hemorrhage followed the introduction of the catheter in two cases. A marked anæsthesia of the glans penis and mucous lining of the urethra was found present in a number of cases; several of the patients assured me that they could hardly feel the instrument passing. The anæsthesia, probably, is the result of the hypertrophy of the mucous membrane, and changes in the terminal corpuscles of the sensory nerves supplying the urethra and glans penis.

In six of the patients examined the prepuce covered the glans, which could easily be uncovered. In two a partial phymosis was present so that the glans could not be uncovered without pain. In one case the phymosis was so nearly complete that considerable difficulty was experienced in introducing an instrument to divide the prepuce. The veins of the scrotum were enlarged (varicocele) in one of the patients examined. No other abnormal conditions of scrotum or testes were noticed beyond an occasional relaxed condition of the former. There is one peculiarity that I have frequently noticed in connection with these cases: a reluctance to have the eyes examined. It seems almost impossible for them to look directly at any one, and when asked to do so they usually avert the head after taking a hasty glance.

The diagnosis during the first stage is by no means always easy, as they will often use every artifice to deceive. The general deportment, loss of memory, fear of impotency, facial twitchings under excitement, an inability to gaze directly at the examiner, and the condition of the genitalia prove valuable aids in confirming a diagnosis. Spots on the bed clothing are no proofs of masturbation, as they are usually the result of involuntary emissions, and may cause groundless suspicions, while the guilty party, using every precaution to prevent even the staining of his body linen, escapes unsuspected.

The diagnosis during the second stage is comparatively easy, especially in the wards of an asylum, where they can be closely watched by day and night.

The frequency with which some of the insane masturbate is truly astonishing. W. P. has been noticed to masturbate six times in twenty-four hours, and a day rarely passes that he is not detected once or oftener, and although greatly demented he has the appearance of enjoying fair general health. Fits of passion with homicidal tendencies are occasionally manifested by these undoers. The prognosis is always unfavorable unless the practice is discontinued. The dangers connected with the vice are frequently under-rated by practitioners of medicine, who to counteract the exaggerated statements of charlatans and popular works designed to excite the fears for a mercenary end, go to the opposite extreme of regarding the practice as harmless. The impaired general health, loss of memory and nervous prostration that accompany the practice if excessive, refutes the truth of all such statements, and prove conclusively that the effects on mind and body are pernicious. Dr. Bucknill, alluding to the effectual operation, writes: "But these miserable helots of sensation who are the usual victims of this despicable vice are not worth the responsibility of even such a thought." The prognosis is not hopeless in all cases however, and undoubted recoveries take place in every large hospital for the insane. Mr. W., aged nineteen years, a confirmed masturbator, was admitted to this hospital in 1876. Masturbated several times a week since fourteen years of age;

parents living and well, and no history of heredity in either branch of his family. His insanity was regarded as due to masturbation by all the medical men that examined him. After several months of treatment he was discharged recovered and has remained well ever since. I was informed by him after recovery, that ever since he commenced taking the medicines, he had no desire to masturbate. I have also been informed since he was discharged that his general health is better than it had been for years prior to his admission.

Diet, exercise and medicines will be separately considered as the principal factors entering into the successful treatment of these unfortunates. The diet should be highly nutritious but not stimulating; the regulation diet of hospitals containing in sufficient quantities all the proximate principles of the body, is well suited to the wants of this class of patients. Stimulants of any kind, alcohol, tobacco, and even coffee should be used very sparingly if at all.

Exercise carried to fatigue I regard as an important element in the treatment, daily out-door employment should be insisted on if practicable. Several patients of this class derived marked benefit last summer while digging a long ditch; the work was laborious, and while it prevented masturbation during the day, it produced such fatigue that they were glad to rest at night. Their minds instead of brooding over their lamentable conditions were pleasantly occupied with their work, and strengthened in proportion to the improvement of the general health. When there is no out-door employment, or the inclemencies of the weather are such as to make it impracticable, daily employment in shops should be enforced. These patients certainly will not be benefited by removal to an asylum if allowed to spend their time in idleness, and such removal will prove an actual harm by increasing the amount of unoccupied time, some of which under ordinary circumstances would have been spent in work or amusement. On the wards they should be closely watched, and it might be advisable to have them occupy a bed with a patient not addicted to the vice who might exert some influence over them. The medicines employed should fulfill the following indications: improve the general health, remove sexual desire and pro-cure sleep. Of the remedies recommended those that afforded the best results were the preparations of iron, the bromides, chloral hydrate and hyoscyamus. Twenty grains of potassium bromide with ten drops of tinct. chloride of iron three times a day, and forty grains of chloral hydrate at bed-time have been followed by marked benefit in a large percentage of cases. Where the disease has progressed to a stage of dementia, all treatment will be in vain. Cauterization of the prepuce has been made use of with indifferent success in a number of cases. Mechanical restraints, as confining the hands and legs, are useless, and stimulate the desire to accomplish what they are intended to prevent, and no restraint unless too barbarous to employ would prove successful.

In conclusion I think I am justified in making the following deductions :

I. That masturbation is an exciting cause of insanity.

II. That in a small percentage, certain physical conditions are present due to the vice, and may prove valuable aids in confirming a diagnosis.

III. That the general health of insane masturbators is always impaired.

IV. That the diagnosis in the first stage usually is difficult, and comparatively easy in the second stage.

V. That the prognosis is always unfavorable unless the practice is discontinued.

VI. That daily exercise carried to fatigue is an important element in the successful treatment of these undoers.

VII. That they are not benefited by removal to an asylum if allowed to spend their time in idleness.

VIII. That certain medicines by improving the general health and removing sexual desire prove successful in some cases.

IX. That cauterization of the prepuce, and physical restraints, as a rule, are impracticable or useless.

Art. IV.—THE ARCHITECTURE AND MECHANISM OF THE BRAIN.

By Edward C. Spitzka.

PRELIMINARY CONSIDERATIONS.

ALL higher manifestations of nerve force have for their organic substratum some one or several of the organs collectively designated as the central nervous system.

If we compare the mental manifestations in a given series of vertebrates on the one hand, and the development of this central nervous system in the same series on the other, we shall find that the higher the mental capacity, the more pronounced is the *differentiation* of the cerebro-spinal axis.

Again, if we trace out the successive evolution of the nervous system in the embryo, we find that the earlier embryos of lower and of higher animals are in this respect almost undistinguishable from each other, and that the human brain presents phases which in their serial succession closely simulate a series of adult brains of lower animals, arranged in their respective order of *complexity*.

Just as it is therefore evident that anatomically the substratum of the human mind has gradually developed from a condition far simpler than that of the brain as found in the adult, so may we infer that physiologically the highest nervous, that is the intellectual manifestations, are based in part on the unconscious registration of an immense phyllogenetic past, projected, as this is, in the anatomical perfection, produced *gradatim*.

The relations of the embryonic to the adult nervous system, in the light of this conclusion, assume a high degree of importance, and this importance is enhanced by the further and practical consideration that a knowledge of the embryonic nervous system bears to the knowledge of the adult nervous system the same relation which the study of the scaffolding of an edifice bears to a study of the edifice itself: it is an almost essential preliminary.

The cerebro-spinal axis of the adult presents us with such a maze of ganglia, such complicated topographical relations, and such a bewildering labyrinth of unilateral and commissural associating strands, and of direct as well as decussated and interrupted projecting tracts, that not only the student of general medicine, but even many of those who devote their attention to neurology, abandon the task of obtaining more than a knowledge of the cerebral outline as hopeless.

To lessen the labor of those who desire to understand at least so much of the cerebral architecture as is necessary for the comprehension of the cerebral mechanism, is the object of these lines. Following the architect's example, we shall endeavor to explain the human brain, by tracing its foundations as presented in the embryo and lower animals, and then following out the progressive building up of the elaborate superstructure which this organ presents in the human adult.

It is not proposed to furnish herewith a complete embryology or comparative anatomy of the brain, as little as it is our purpose to discuss histological and physiological questions *as such.* These pages, as indicated in their title, have no other unity in their plan than the accommodation of the physiological theory to the anatomical basis. Wherever the results of collateral science have appeared to us as calculated to assist in the clearer demonstration of the cerebro-spinal architecture, we have not hesitated to employ them. At the same time we have avoided these themes, where their introduction would have proved at best but a useless ballast.

§ 1. The characteristic attributes of every animated being are sensation and motion. Sensation consists in the molecular changes induced by outward influences acting on the animal; motion consists in other molecular changes which result in any alteration of the animal's position or form. In the simplest, as well as in the highest animals, motion is dependent on sensation; without sensation to guide it, motion would be an entirely useless property. It is evident that sensory influences must precede every *orderly* motor reaction!

Since both sensation and motion are ultimately reducible to molecular oscillations, the reaction of motion on the sensory influence must be considered as a transmission of molecular oscillations *from* the sensory *to* the motor end organs. Where the animal molecules are diffusely and homogeneously mingled, so that sensory and motor end organs are not differentiated, it is easy to see that molecular oscillations must be transmitted from one to the other by the mere contiguity of the tissue molecules.

The course of progressive development is to make the animal as independent of its immediate surroundings as possible, that is, to increase its *freedom of choice.*

While the amœba will go on forever sending out its sarcode-processes and encircling floating objects, as soon as these come in contact with it, a higher animal will, under the same circumstances, have the choice of two alternatives—alternatives which the simple organization of the amœba is incapable of holding, the amœba being a blind slave to the immediate juxtaposition of its sensory and motor elements.

§ 2. The freedom of choice implies that the current passing from the sensory to the motor elements shall pass through an *intercalated medium.* This medium, which we will for the present *assume* to be capable of either *liberating* or *inhibiting currents*, is the nervous system.

§ 3. The nervous system follows in its structure the same plan exhibited in the bodily shape. It is built on the radiate plan in the echinodermata, it is distinctly segmental in the arthropoda, and in the vertebrata consists of an axial tube, which is also segmental, but less markedly so than in the arthropoda.

Let us take an ideal animal, and suppose it composed of a number of segments arranged end to end, giving it a worm-like shape. We will suppose each segment to be composed of sensory and motor elements, and a nervous system consisting of a simple arc, that is, a ganglion with an afferent fibre *from* the sensory, and an efferent fibre *to* the motor apparatus. If this were the sum total of the nervous system, each segment would appreciate sensation and enact movements entirely independent of the whole animal, and if impressions of different kinds were brought to bear on different segments, these segments would act so as to interfere with each other!

The organic necessity under which the animal is placed, demands harmony of action, and to produce this, it is necessary that the ganglia of different segments shall be in some sort of union. This union is effected by the *associating tracts.*

Through the tracts which connect the ganglion with the sensory and motor periphery of the same segment, the latter are projected in the ganglion; these are, therefore, its *projecting tracts.*

The basis of the most, as of the less complex nervous systems, is a series of ganglia, with projecting and associating fibres.

§4. As the extensity of a given force depends upon the mass of molecules, whose oscillation determines that force, it is clear that the larger the segment, in other words, the more developed its sensory and motor organs, the larger must be the ganglion of that segment.

From this fact we derive the first great law of the cerebral architecture, namely, *a ganglion* (*centre*) *follows in development the development of the periphery, which is projected in that ganglion.*

The lumbar enlargement is more marked in animals possessing powerful posterior extremities (man, kangaroo) than in those possessing weak or rudimentary ones (bat, porpoise). The cervical enlargement is proportionately larger in the bat, with its anterior extremities over-developed, and in the mole (for a similar reason), than in the dog and rabbit. The oculomotor and trochlearis nuclei are almost absent in the *pipistrella* bat, and entirely so in the mole, since the eyes of the former are poorly developed, and those of the latter rudimentary. The lower facial nucleus of the elephant follows the hypertrophy of the facial muscles (trunk); the hypoglossal nucleus in the seal is reduced, just as the tongue is limited in motion. The anterior tubercles of the corpora quadrigemina are atrophic in the bat and mole for the same reason assigned in the case of the oculo-motor nuclei; and in the land turtles the extreme atrophy of the parietal muscles in the dorsal region is accompanied by a greater diminution in the area of, and number of cells in the dorsal grey matter, than in any other animal. *Per contra*, in the axolotl and other urodela, as well as in the apodal lacertians (pseudopus) and snakes (anaconda, boa, rattlesnake), the cervical and lumbar enlargements are either scarcely, or not at all perceptible, just as the limbs are absent or insignificant.

§5. The ancestral vertebrate was a swimming animal, and its locomotion took place in the direction of the long axis. It was built upon the segmental type, and the nervous system may, for the clearer demonstration of what is to follow, be considered as composed merely of a series of segmental ganglia, and projecting as well as associating tracts, such as were mentioned above (§3). As the segments in this ideal progenitor were originally all alike, the segmental ganglia must also be considered as alike, and none of them as preponderating over the other.

It alters nothing of this representation that even the simplest living vertebrates possess an unsegmented nervous tube. The ganglionic segments are to be considered as having fused typographically, while physiologically they have maintained their segmental distinctness. With all this, the nervous axis, even in high animals, exhibits a segmental *derivation.* In some fish (orthagoriscus, and our common gurnard) the spinal cord is composed of a series of enlargements, from each of which a pair of nerves arise; and all histologists must have remarked, in preparing sections from the human spinal cord, how a series of sections will be poor in ganglionic bodies, the next will be rich in these; again a barren field will follow, and so on in a quite regular alternation. The intervertebral ganglia, derived as they are from the medullary tube, maintain their segmental individuality throughout.

Now as this creature progressed through the water, the *anterior segments* were those which *first* encountered the foreign objects floating in the same medium; it was for these segments to determine whether these objects were inimical or indifferent or desirable to its individual ends! In the course of countless ages and through natural selection, the sensory periphery therefore underwent a high specialization *here*, certain of the end organs thus appear specially destined to appreciate the quality of the medium inhabited (olfactory), others to receive the rays of light, others certain oscillations of the surroundings (auditory), others finally, the strictly tactile organs, undergo an elaboration as such (tentacles, cirrhi).

But not only did certain special sense peripheries develop on the anterior or head segments, peculiar and important motor contrivances also became necessary. In the first place, since the head end was the first to encounter the prey to be captured or the enemy to be warded off, it was provided with powerful mandibular muscles, and muscles of deglutition.

Since in a water inhabitant, the respiratory medium was a fluid rushing in at the mouth, as the creature progressed through the water, the anterior or head part of the digestive tract underwent a modification resulting in the formation of gills at the very point where the respiratory medium was not rendered impure, as it would have been if it had had to pass through the whole length of the animal before becoming utilized. And since the object of the respiratory apparatus was to bring the circulating fluids within the animal in contact with the gases contained in the respired medium, what more natural than that the circulatory centre, the heart,* should be also situated in the head end? The nearer the gills the heart, the less force is lost by the branchial blood-current.

Bearing in mind our first law announced, we perceive in this differentiation of the head *periphery*, the reason for the great development of the head *centre*. The eye, the *regio olfactoria*, the organ of Corti, the semi-circular canals, the gustatory papillæ, the muscles of deglutition, of ocular motion, of the maxillaries, as well as the great centres of circulation and respiration, are all concentrated on the first few segments of the body, and it is for the ganglia of these segments to increase correspondingly. Thus it is that the first ganglia preponderate over all the others as a *Brain*. And thus it is that the brain shows such complex relations in contrast with the relative uniformity found throughout the spinal cord.

§ 6. But it was not only the *ganglia* of olfaction, smell, taste, mastication, respiration and circulation that underwent such an increase in dimensions; superior as they were in functional importance to all the other ganglia, and exerting therefore a control over these others, it followed that the *associating tracts* joining the cerebral and spinal centres predominated over the associating tracts joining the spinal centres to their fellows.

The higher a brain in the scale, the more complex, the more voluminous also the tracts joining the brain and cord in functional union.

We have here a fundamental law of the cerebral mechanism: *The highest functions do not reside in any special centre, but in the functional union of several centres through associating tracts.*

Before adding further explanations of this law, and in order to understand its exact application, other features of the cerebro-spinal architecture dependent on its embryological development, must be understood as a preliminary.

It must not be forgotten that while the embryonic developments in their main features correspond to the ideal progression of the vertebrate series in evolution, that there are many modifications for special purposes, which forbid the regarding of the embryonic as an exact reproduction of the phyllogenetic development. On the other hand we should not fall into the error which has found an expression in many current works, that the development of nervous centres necessarily follows in the order of their functional rank. In a general way this is true, but a few months before birth the spinal cord is as far from perfection as several higher centres.

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* In embryos and lower vertebrates the heart is in the cephalic and cervical regions.

Embryological development, in short, follows phyllogenetic and physiological generalizations in their main features only, and is not an exact picture of either.

§ 7. Chemical analysis* shows that the ovum contains the same protein compounds which are characteristic of the nervous tissues. But while in the adult, these compounds are concentrated in certain depots and so disposed as to wield an orderly and methodic control over the other portions of the organism, they are in the ovum uniformly diffused throughout its substance.

§ 8. To this simple condition of the ovuline germ, there is an analogue in the lowest forms of the protozoa. The amœba is composed of as uniform a substance as the holoblastic egg, and just as each portion of the amœba is like to every other portion, so does each portion share all the functions of the entire animal. Any and every part of this simple creature is endowed with motion and sensation, besides the more purely vegetative functions.

Here evidently the nervous and non-nervous constituents must be chaotically intermingled, as compared with the degree of separation attained in higher animals.

From the optical and physiological similarity of the ovuline to the amœboid protoplasm we are justified in inferring that the former like the latter contains representatives of the nervous tissues, uniformly and diffusely distributed throughout its mass.

The correctness of this analogy is borne out by the fact that the impregnated as well as in a lesser degree the unimpregnated germs manifest *amœboid* movements. The germ of the trout, on account of its size and transparency, presents the finest illustration of these movements with which we are acquainted; it sends forth long processes, spreading out flat over the nutritive yolk, or collects itself into a spherical clump, all the while exhibiting flowing motions of its protoplasmic granules so distinct as to be visible even under a low power.

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* Hoppe-Seyler, *Med.-Chemische Untersuchungen*, 1867.

§ 9. This crude condition of the germ merges into a higher one, even before the act of impregnation takes place. Preceding and accompanying this act, groupings of the finer and coarser grained protoplasms into separate formations have been observed* with other changes, which although they are not as yet susceptible of a clear interpretation, must be considered as preparatory stages of the more profound differentiation which inaugurates segmentation.

§ 10. Segmentation begins by the cleavage of the germ into two *unequal*† hemispheres. Both alike undergo repeated subdivisions until the well-known mulberry appearance ensues. It is noteworthy that the *larger* hemisphere which furnishes the substratum of the central nervous system (as we shall see) segments much more rapidly than the lesser. From this it naturally results that the derivatives from the larger hemisphere must spread out over and finally surround the derivatives of the other, and as these latter have by a central separation of their elements, become arranged like a sac around a cavity, and are in their turn lined without by the former, we have the *now vesicular* ovum composed of two concentric hollow spheres. These are known from their relative position as the ectoderm and the entoderm.

§ 11. The ectoderm, *chiefly devoted to the building up of the nervous system* and the organs of special sense, is derived from the larger hemisphere referred to above, and allusion has been made to the fact as being of some significance, that the primordial substratum of the nervous axis exhibits a far greater activity in the course of segmentation, than the substratum of most other organs. *It seems as if the higher functional potentiality of the nervous tissues were heralded in this more intense segmentative energy.*

§ 12. A portion of the vesicular ovum is remarked as being the seat of those changes which are most intimately related to the development of the embryo, it is therefore known as the *area embryonica;* within its limits the entoderm separates into two layers, of which the deepest is known as the hypoblast, and the other, nearer the surface, as the mesoblast. The latter is just beneath the ectoderm, which for purposes of nomenclatural uniformity we shall henceforth designate as the epiblast. There are therefore three strata in the *area*, which at first are mere membranous expansions, overlying each other; but soon a thickening occurs in the outer layer or epiblast, which forms the starting-point of the numerous and intricate changes culminating in the formation of the brain and cord.

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* By Eimer, Romiti, Ed. van Beneden, van Bambeke, Balbiani, Shenk, Törok, Goette, Hertwig and Fol.

† Ed. van Beneden.

§ 13. This thickening is in the axis of the future embryo, and is due to a local increase in size and number of the *epithelium-like* cells of the epiblast; it is the *primitive medullary lamina.* In obedience to certain laws of growth which it does not lie within our province to discuss here, this lamina becomes bent parallel to its long axis, like, to use a homely comparison, a long strip of tin would be rolled to form a gutter. The bending process is continued, so that the gutter becomes a pipe; in other words the lamina which was gradually transformed into the *medullary groove* is now a *medullary tube.*

Although embryologists have long corrected the old error that the *primitive streak* and *primitive groove* are the precursors of the medullary tube, yet several works on the central nervous system still cling to this erroneous and antiquated notion. This may serve to excuse a special reference to the fact (which reference ought to be superfluous) that the primitive groove or furrow is a temporary appearance, early and entirely obliterated, of no relation to the nervous system, and having a signification only for the zoologist, as demonstrating the existence of a *gastrula* stage in vertebrates.

§ 14. From the walls of this tube are developed the nervous tissues of the central nervous axis. The canal itself is the forerunner of the central canal of the spinal cord as well as of the *true* cerebral ventricles.

§ 15. It is by local-differentiation, by preponderating growth of this, or atrophy of that part of the tube, by the mechanical influence of surrounding protective capsules, or the as yet undissolved connections with other embryonic structures, that the manifold differences of various sections of the cerebro-spinal axis are to be explained.

However complex the cerebrum, however numerous and apparently distinct from their surroundings the basilar ganglia and the nerve nuclei, however intricate the course of the nervous tracts of the isthmus in their primordial origin, they are no more complex and no more differentiated than are the nervous elements of the cord. From the olfactory lobe to the conus terminalis all nervous tissue is originally represented by a simple *epithelial tube.*

§ 16. We have already (§ 4–5) in speaking of the ideal ancestral vertebrate referred to the close connection existing between the nerve centre and periphery of each animal segment. The same close connection is maintained in the embryo of the highest animals and this is the foundation of one of the most important modifications of the central nervous axis.

It is clear that the epiblast thickening when it becomes gradually indented and rolled into a tube, must retain its connection with the *parent epiblast* longest at its *dorsal aspect*, while the *ventral aspect* is driven down among the subdivisions of the *mesoblast.* Now as delicate fibrils begin to unite the medullary tube with the surrounding parts, and following the law of propinquity, connect each part of the medullary tube with those neighboring parts which are *nearest* to it, it results that the dorsal aspect is connected through the primitive nerve fibrils with the parent epiblast, the ventral (and lateral) aspect with the mesoblast. The epiblast develops the *sensory*, the mesoblast develops the *motor* end organs. Hence we have a fundamental differentiation of the nerve axis here, which is destined to influence the architecture of the brain and cord throughout life. *The dorsal (posterior) portion of the nervous axis is connected with sensory organs, the ventral (anterior and lateral) portion is connected with muscles.*

The accompanying diagrams (schematized from sections transverse to the axis of rabbit embryos of different stages) may serve to elucidate this relation. In all the epiblast is represented as composed of black quincunx spots, the mesoblast as striated, the hypoblast by open cubes.

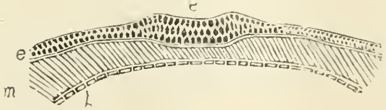

Fig. 1.

Figure 1 represents the earlier stage; here the three layers, epiblast, mesoblast, and hypoblast overlie each other as flat expansions; the epiblast (e) exhibits a medial thickening, the medullary lamina (ə).

In a later stage (Fig. 2) the whole dorsal contour of the embryo becomes more convex; the medullary lamina becomes metamorphosed into the medullary groove (e); the dorsal aspect of the medullary groove can be seen to be continuous with the parent epiblast, and this is the future sensory segment of the medullary tube. The other or ventral part of the medullary lamina has moved to a position intermediate between the axial chorda and the protovertebræ (p), from which the voluntary muscles are derived.

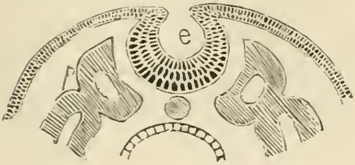

Fig. 2.

The mesoblast shows three primary subdivisions: one medial (the chorda) and two lateral parts. The lateral parts divide in turn into the protovertebræ (better termed segments, Götte, Entwicklûng der Unke) which lie nearest the medullary tube, and the lateral laminæ (segmental laminæ, Götte, *ibidem*). The cavity represented in the diagram as situated within the protovertebræ becomes obliterated; it is an isolated division of the slit observed between the inner and outer lamellæ of the lateral laminæ. This slit is the cœloma, or to use the more expressive polysyllable, the branchio-pleuro-pericardio-peritoneal cavity.

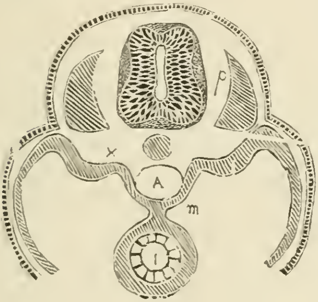

Fig. 3.

The lamella on its outer side does not interest us here; the lamella on the inner side, as shown in Fig. 3, forms the involuntary muscles of the aorta (A), thence the inner lamellæ of opposite sides join to form the mesentery (m) and then surround the hypoblast, which has closed to form the *enteric epithelial tube* as the muscular tunic of the viscera.

The cross-piece (x) uniting the outer and inner lamellæ, is the basis of the urogenital system.

Now it is easy to see that *from the beginning* the dorsal part of the medullary tube, when its fibrillæ sought peripheral connections (according to Hensen it is doubtful whether protoplasmic connections of the cells formed after segmentation are *ever* absent) must naturally have connected with the parent epiblast (Hornsinnesblatt), or the *sensory* and epithelial layer of the integument. And also that the ventral and lateral aspect must connect with the voluntary and involuntary motors. The subsequent union of the concentrated roots is a matter of purely mechanical moment, and does not alter this relation.

At the point where the parent epiblast was longest in connection with the medullary tube, the ganglia of the *posterior* roots (gasserian and intervertebral) are developed (these are not represented). It seems that cells analogous to those of these ganglia are *contained permanently within the nervous system, at two points where the mechanical factors which determine the separation of the intervertebral ganglia are absent or imperfectly exercised*, that is in the anterior segments of the medullary tube; to us it has always seemed as if the spherical nerve cells of the dorsal grey of the aqueduct and the cells of the *locus cærulen* were of this kind.

It may be well to state here that originally the nerve roots are diffuse and uniformly scattered around the periphery of the medullary tube, and that condensations at the root-zones occur only secondarily.

The fibrillæ which compose the future white covering of the cellular layer of the tube, show, according to Hensen,* three fundamental systems, a vertical and a radiatory, besides a circular. With advancing development the radiatory concentrates itself and develops at the expense of the longitudinal at the root-zones and raphes; the vertical develops at the expense of the radiatory *between* the root-zones, that is, in the white columns. The circular attains an important degree of development in the arciform fibres, the restiform decussation, trapezium and pons.

We have satisfied ourselves that this view of Hensen is correct, and agree with that author that the employment of the routine Canada balsam and damar mounting of specimens, renders many of the finer points in histogenesis invisible. But even in balsam specimens we have been able to see the multiplicity of the primitive rootlets (rabbit and batrachian embryos); hæmatoxylin specimens from chicks ranging from the fourth to the twelfth day of incubation finely show the gradual replacement of the uniform reticular arrangement of the fibrils, by the permanent fasciculi, at the expense of the outermost line of the proliferating cells of the tube.

It appears to us that just as the general differentiation of a motor and sensory district is explicable by the law of propinquity, that finer differentiations can be similarly traced, up to a certain point. The ventral muscles of the trunk and the flexors of the extremities would have their nuclei nearer the anterior commissure; those of the back and the extensors of the extremities nearer the so-called lateral cornu. Türck (Sitz, *Bericht der Wiener Akad.*, Band VI.) has proven a corresponding arrangement of the sensory provinces, referrable to the same embryological propinquity.

§ 17. As yet the medullary tube consists of nothing but more or less cubical and cylindrical cells, therefore we cannot speak of grey or of white substance: *both* are yet to be formed from these cells.

§ 18. Immediately surrounding the lumen of the medullary canal, the cells retain their epithelial character, and become in the adult the epithelium of the ventricular and spinal *ependymas.*

§ 19. Further outwards while the cells undergo proliferation, their protoplasm loses its contour; the cell bodies run together into a common mass, and the medullary tube exhibits the appearance of a uniform blastema, containing a large number of nuclei. These nuclei are the centres of formation for the *ganglionic bodies* commonly termed nerve cells.

§ 20. At the same time, the fused protoplasm of the cells develops delicate fibrils. These fibrils soon extend beyond the limits of the cells, so that we now have an inner portion of the tube composed of the blastema and apparently free nuclei, and an outer composed of delicate fibrils which have developed from the blastema, and are more or less in connection with it. In very early embryos this fibrillary layer is an almost undemonstrable mere outer lining of the medullary tube, but it grows rapidly, and by subsequent nutritive changes, its fibrils become enveloped by myelin, giving it the characteristic appearance of *white* substance. Within the area of the nucleated blastema, there are also fibrils; but here they do not as a rule develop as differentiated a sheath as the fibrils in the white substance.

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* Hensen, "Entwicklung des Kaninchens."

§ 21. Now the central nervous system contains two great categories of substances. The one which lies nearest the lumen of the medullary canal has been proven by experiment to be the seat of those molecular oscillations which determine the reception of impressions, and the motor reaction on these impressions. The substance of which it is composed usually exhibits itself in certain accumulations known as ganglia, for which reason we shall speak of it as *ganglionic substance.* It is indifferent for the first stages of this inquiry what the morphological arrangement of this ganglionic matter may be, whether it is arranged in cornua as in the cord, in nuclei as in the medulla, in scattered and diffused formations as in the entire isthmus, in large nodular accumulations, or surface expansions, as in the basi cerebral ganglia and the cortical grey substance; ganglionic matter has these fundamental physiological attributes, that it receives impressions and generates motor impulses.

§ 22. The second category is one without which the first, notwithstanding its high organization, would be a mere mass of functionless living matter. It has for its object the uniting of the ganglionic substance with the other tissues of the body. If this tissue is a sensory organ, it conveys the impressions received by this organ *to* the ganglionic substance; if this tissue is a muscle, it conveys motor impulses *from* the ganglionic substance to the muscle. In both instances it plays the rôle of a conductor; and, therefore, we speak of it as the *conducting substance.*

Anatomically, the ganglionic and conducting substances are not sharply demarcated. While those conducting strands, which for their more thorough isolation are provided with a myelin sheath, are grouped together in distinct fasciculi, the naked or grey fibres run directly into the grey substance. So that while the white substance is purely conducting, the grey is *both ganglionic and conducting.* In many localities even white fibres encroach on the ganglionic substance, as in the strands found in the fourth layer of the cerebral cortex, the white layer of the occipital cortex, and many other localities.

§ 23. It is an error to believe that the evolution of nerve force implies the existence of distinct fibres and of so-called nerve cells. Any protoplasmic accumulation of the chemical composition of ganglionic tissue, placed under the same influences as the latter is placed under in the living animal, is capable of developing nerve force. In the lower forms of animal life a uniform blastema with or without nuclei, and entirely devoid of nerve cells, is frequently all that represents a ganglion, and the afferent and efferent strands connected with such a simple structure are composed of an infinite number of granules which show no fibrillary arrangement.

Now as these lower animals are capable of performing comparatively complicated acts, and as the performance of complicated acts implies that the nerve machinery shall exhibit a certain constancy in its action, it is evident that there must be something in the *molecular arrangements* which forces the nerve currents to travel in given paths—paths which are defined and permanent, physiologically speaking, though undistinguishable to the anatomist!

§ 24. With the greater complexity of peripheral and intrinsic connections which the nerve axis presents in higher animals, the molecules begin to present that definite arrangement to the eye whose existence in lower animals was impalpable, except to physiological deduction. *The increased perfection of the nervous system is marked by a progressive tendency to isolation of the conducting strands.* We have the granules grouping themselves in granule-rows, or fibrils; and finally we have these fibrils surrounded by an isolating sheath.

At first in the embryo (§ 20) we have the conducting substance grouped in fibrils, which are connected with the periphery on the one hand, and terminate in the blastema, which contains the nuclei of the originally distinct epithelia of the medullary tube, on the other.

§ 25. Radiating inwards through the substance of this tube, these fibrils make their way by creeping between, and meandering amongst, the free nuclei. At many points it can be clearly perceived that these fibrils form distinct groups around one or other of the free nuclei, and in doing so they include in their meshes a quantity of the blastema which surrounded the nucleus (Fig. 4). In this manner the ganglionic bodies are formed; a ganglionic body, therefore, consists of a nucleus, a certain amount of protoplasm around this nucleus, and of a greater or lesser number of fibrils which pass through, or at least penetrate into this protoplasm.

§ 26. The ganglionic corpuscle is hence a very complex body, and is not a cell in the proper sense of that term; but tradition has distinguished it as the *nerve* or *ganglion cell*, and as these terms will probably be adhered to in the English language to the end of time, we shall also use them, for the better comprehension of the reader, with the reservation mentioned.

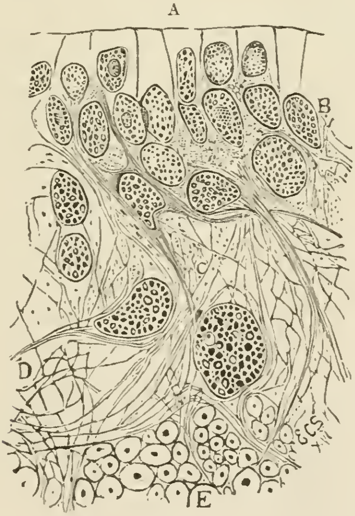

Fig. 4.

Section from the floor of the fourth ventricle at its posterior part (*menobranchus lateralis*, or American proteus); A, epithelium of ventricular floor, showing the large granular nuclei of the epithelia; B, nuclei, showing a transition to the nuclei of nerve cells; C, fibrillæ, with scattered protoplasm; D, fibrils grouped into the process of a nerve cell, directed to raphe; E, white substance. × 350 diameters from author's specimen.

§ 27. From the fact that nerve cells are absent in very low animals, and that they become better and better developed and more and more numerous as we pass upward in the animal scale, but one inference can be drawn, namely, that *the nerve cell is the ultimate functional centre of the nervous system in man and other higher animals.*

It is necessary to insist on this fact, long ago accepted as a foregone conclusion, but recently attacked or questioned by some of our most eminent investigators. Some have claimed that the neuroglia is the real central field of nerve force, and that the nerve cell is merely a passage point for conducting fibrils, which are thus brought into a definite arrangement prior to their peripheral distribution. To this it can be answered that the neuroglia is relatively and absolutely present in greater quantity in lower animals than in man. And while we admit, and ourselves insist, that one object of the nerve cell is to bring numerous fibrils into definite groups, yet that this is not the only object of this elaborate body, is shown by the presence of a nucleus *unconnected* with the nerve fibrillæ, and of protoplasm scattered (however sparsely) among the fibrils. Nature has not placed these chemically highly complex bodies here without an object! The nerve cell is anatomically an elaboration out of the crude blastema, and just so physiologically its importance rises far above that of its mother bed, which latter in higher animals documents its waning physiological importance by simulating the character of a connective tissue. The neuroglia may hence be regarded as a part of the original simple ganglion, which retains its functional capacity in such a rudimentary form as practically to be of no importance in the mammalia, while the nerve cell is the part which usurped most of the functional material, and rose to the dignity of an individual centre.

§ 28. Careful anatomical research has shown that the nerve cell is provided on the one hand with peripheral connections, on the other with connections uniting it directly or indirectly with its fellows. Each single nerve cell exhibits therefore first ganglionic substance, second, connections *projecting* the motor or sensory periphery, and third, connections *associating* it with similar bodies. *The nerve cell is therefore a physiological unit, being provided with all the anatomical attributes which we predicated for the segmental ganglion* (§ 3).

§ 29. If we examine the nerve cells of a given cerebral ganglion, *known* to be the seat of a function or combination of functions which rises in extensity in the ascending animal series, we shall find that the processes with which the cell is provided are few and simple in lower animals, and become more and more numerous as we pass up in the animal scale.

The cells in the anterior spinal cornu of the frog are very rich in processes, those of the salamander, and still more so those of the siren are therein poor; the spinal co-ordination of the frog is correspondingly higher than that of the urodela. The nerve cell of the cerebral cortex is a free nucleus in the menobranchus, bipolar in the amphiuma (Schmidt), has but few more processes in the scaly reptiles, fewer in the rabbit than in the dog, in the dog than in the ape, and in the ape than in man. (Herbert Major states in his paper on the cortex of a cynocephalus baboon, that he could discover no other difference between the nerve pyramids of the human and simian cortex than the lesser richness in processes of the latter. We can confirm this observation for macacus and cebus; in the chimpanzee we could discover no difference, taking into account that the staining was imperfect.) The proteus, amphiuma, reptile, rabbit, dog, ape and man occupy with regard to the respective number of processes appended to the cortical cell the *same* order which they occupy in the intellectual series!

Here we perceive that the nerve cell, following the law which we have announced for the *entire nervous system*, *gains in functional dignity with the increase of its associations.* We are justified in anticipating that where a given nerve centre varies in functional complexity in different animals, the cells composing it will show a parallel and related variation in the number of cell processes. The further inference will suggest itself to the reader, that where in a large series of animals, whether their motor co-ordinations be relatively sluggish or quick, whether their sensation be ready or obtuse, their intellect low or high, a given group of cells shows no variation in the number of its processes, that group of cells is not very likely to be in relation with voluntary motion or "conscious" sensation. By exclusion we should infer such a cell group to possess a trophic signification, for the trophic functions show the least variation in the animal range!

The application of this inference in the anatomy of the grey matter of the aqueduct and third ventricle will be discussed in its proper place. It is one of the numerous inferences which, theoretical as they are, constitute our sole guide in the obscurer fields of cerebral anatomy; their basis is highly logical, and although they may forever remain incapable of exact demonstration, yet all collateral facts are in their favor, while none can be urged against them. The trophic functions are more marked in the trigeminus than in any other cranial nerve, and this is the very nerve two of whose nuclei exhibit round cells similar to those of the intervertebral ganglia, and like these show no variation whatever in the entire mammalian series.

§ 30. The ganglion cells are all without exception at this early stage grouped around the lumen of the medullary canal. Consequently we have a continuous tubular cell column, extending from the anterior to the posterior extremity of the central nervous system, that is, from the anterior limits of the third ventricle to the filum terminale. The cerebro-spinal nerve fibres of the successive corporal segments terminate in topographically corresponding portions of this tube.

The primitive central nervous projection field for the peripheral end organs is therefore to be sought in the CENTRAL TUBULAR GREY MATTER, *that is in the grey matter of the spinal cord, the grey matter of the floor of the fourth ventricle, the grey matter surrounding the sylvian aqueduct and that lining the sides and floor of the third ventricle.*

Since the grey or ganglionic matter in which a spinal or a cranial nerve terminates, is often designated the nucleus of that nerve, the central tubular grey matter is also known as the *nuclear formation.* But not all so-called nuclei are equivalent to the central tubular grey matter in a strict sense: the term should be limited to the grey matter situated around the lumina mentioned, and not attached to the internodial nuclei of certain nerve strands.

The central tubular grey matter shows a tendency to nuclear differentation in all higher animals, and from this only such parts of the axis are exempt as have by becoming supplanted through secondarily established higher ganglionic depots, lost their functional importance. Thus the floor of the third ventricle over the chiasm, known as Meynert's basal optic ganglion, is the atrophic remnant of the primitive optic centre. From this point the pedicles of the ocular vesicle originated, and in the lowest fish the optic nerves terminate here (myxine). These relations will be discussed in their proper place, and their signification pointed out.

§ 31. The ganglia or nuclei of the central tubular grey matter are arranged in symmetrical groups. These groups on opposite sides of the median line are brought into functional association through fibres which cross the median line; this series of transverse fibres developed throughout the cerebro-spinal axis, constitutes the *transverse associating system of the primitive cerebro-spinal centres, i. e., those of the central grey.*

§ 32. The centres situated on different planes of the cerebro-spinal axis, but on the same side of the median line, are also united with each other; those situated in immediate contact, by the fibrillæ of the grey substance itself, those situated at greater distances are connected by fibres which leave one centre to run through the white substance longitudinally, and enter the other lower or higher centre associated by its means. This is the *longitudinal associating system of the primitive cerebro-spinal centres.*

§ 33. The nervous system which we are now constructing, thus far consists of (a) a bilateral series of segmental nuclei, constituting the central tubular grey matter; (b) of projecting fibres connecting these nuclei with the sensory and motor periphery, known as the peripheral nerves; (c) of transverse associating fibres uniting the bilateral nuclei of the same segment; (d) of longitudinal strands of lesser and greater length uniting variously distant nuclei of different segments, but of the same side.

§ 34. In accordance with the principles enunciated in paragraphs five and six, the segmental ganglia of the head segments undergo a more massive development, and owing to their greater complexity of function develop more powerful association strands than those of the spinal region. Leaving out of consideration for the present everything beyond the third ventricle, we find that the *medulla oblongata* constitutes a hypertrophied segment of the spinal cord, and that the longitudinal associating fibres outweigh in *number* and *length* those of any other spinal segment.

The experiments of physiologists have shown,* that if a sensory irritation of a given spinal nucleus is kept up after having produced a reflex movement in the same segment, if there is any reaction beyond the plane of that segment at all, it is not in the next or succeeding planes, but in the *medulla oblongata.* The motor reaction then manifests itself in laughing, crying or deglutitory spasms, and if the irritation is of the severest kind, epileptic or tetanic spasms in addition.

Now the occurrence of laughing, crying or deglutitory spasms could be easily understood if we imagined the molecular oscillation induced by the irritation to travel along the associating

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* Pflüger, "Ueber die sensorischen Functionen des Ruckenmarks." Berlin, 1853.

tracts from the given spinal segment to the nuclei of the medulla oblongata. For in the medulla we have the nerve nuclei that preside over the facial, laryngeal and pharyngeal muscles. It is not so easy to understand how tetanus and epilepsy, that is spasms consisting in movements whose *direct* projection is *not* in the medulla oblongata, but in the cord, are produced by irritation of the former.

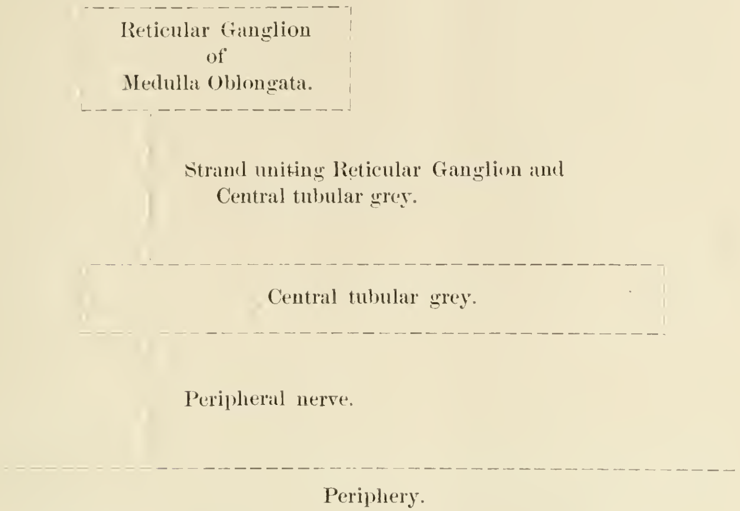
There are scattered groups of nerve-cells in the medulla oblongata, which have mostly either no demonstrable connection with the nerve nuclei, or are positively known to be connected with the longitudinal associating strands. By logical exclusion we are more than justified in considering these cells as representing a presiding centre over the entire spinal system. No spinal centre exerts any influence even remotely as pronounced as that of the medulla oblongata on the *entire* cord.

This applies to man and the mammalia; that the elaboration of the medullary centre was as gradual a process as that of other higher differentiations is illustrated by the case of the frog, where the medulla has acquired the faculty of producing general spasms (it does not according to our experience possess this power in the perennibranchiate urodela) while the cord itself retains this property also, hence here the predominance of the medulla is not as marked as in the mammalia.

That the medulla relatively and secondarily loses in importance in the highest mammals alters nothing of the conclusions of this paragraph, as will be shown.

§ 35. If distal as well as direct irritation of the medulla can equally produce spasms of all the muscles (voluntary and vasomotor) of the body, and if sensory irritation of *any* part of the trunk and extremities can provoke the spasms originating in the medulla, it necessarily follows that all *the sensory and motor end organs projected in the grey matter of the spinal cord must be also projected in the reticular ganglion of the medulla oblongata.* This projection is not an immediate projection such as that established by the termination of the peripheral nerves in the central tubular grey matter, but mediate *through* the central tubular grey.

Accordingly this projection takes place through three elements which are arranged in anatomical and physiological continuity. At one end we have the periphery, at the other the reticular ganglion of the medulla; the connection is established by (a), the spinal nerves, (b) the central tubular grey matter, and (c) the longitudinal associating tracts joining the spinal and medullary grey.


Reticular Ganglion of Medulla Oblongata.
Strand uniting Reticular Ganglion and Central tubular grey.
Central tubular grey.
Peripheral nerve.
Periphery.

Here the ASSOCIATING *fasciculus becomes a part of the* PROJECTING *system.*

§ 36. We have stated (30) that the primitive projection field for the peripheral end organs is the central tubular grey matter, and that the primitive projection takes place through the peripheral nerves. To systematize our explanation of the cerebral architecture, we shall distinguish the central tubular grey matter as the *first* GANGLIONIC CATEGORY, the peripheral nerves as the *first* PROJECTION TRACT, both together constitute a *projection system of the first order.*

The reticular ganglion of the oblongata is not in the adult a part of the central tubular grey matter, but has, though originally developing from it in the embryo become ultimately isolated from its mother bed. It constitutes a *second ganglionic category*, and the association fibres bringing it in functional union with the spinal grey (first category) in lower animals, and shown to have assumed the position of projection fibres in the higher, constitute a *second projection tract;* both together are a second projection system.

The first projection system is perfect in itself, as far as it goes; the second is a supplanted system, and would be imperfect if it did not, so to speak, stand on the shoulders of the first.

Our classification differs from that proposed by Meynert ("Beiträge zur Kenntniss der centralen Projection der Sinnesoberflächen," 1869; Stricker, *Gewebelehre*, II., on the brain of the mammalia). This author, the founder of the projection theory, and as yet the only one who has ventured with any degree of accompanying success to bring cerebral physiology to an anatomical basis, speaks of the central tubular grey as the third category, the great ganglia as the second, and the cortex as the first.

We hold that just as the physiologist must study the reflex processes before studying the automatic and psychical ones, so the lowest and reflex centres must take precedence of the higher in the anatomical delineation. The central tubular grey is the *first* formed, and in the amphioxus is the *only* category of grey matter in the central nervous system. Now assuredly it would be very confusing to the student to speak of the third category in the embryo and the amphioxus when a first and second category are in the former not yet isolated and in the latter permanently absent.

We also differ from that ingenious author in erecting a special category for the accommodation of the *reticular ganglion.* To our knowledge no author has yet given a name to the scattered grey matter of the medulla oblongata, although it has been very accurately described by Clarke, Dean, Meynert, Stilling and others. Its importance inferentially is great, and anatomically it is (though its cells are scattered diffusely *as a rule*) a large ganglion with numerous multipolar cells of all sizes, many of them are of gigantic dimensions and sometimes exceed the so-called motor cells (which they simulate in shape) of the lumbar enlargement in size. Scattered in the "reticular substance" of the medulla from the upper end of the fourth ventricle to the pyramidal decussation they merit the collective designation of a *reticular ganglion*, as we shall develop further on.

While we follow in the main the principles maintained by Meynert, we offer a different classification, one which has physiology, embryology and comparative anatomy in its favor. It remains for the reader to decide whether it possesses the same degree of clearness which attaches to Meynert's excellent plan. Meynert made but three categories (exclusive of the cerebellar grey), we see ourselves compelled to make more, thinking our system to necessitate less bold theorization and generalization, while it is more in accord with anatomy than the *schema* of Meynert. Wundt (*Psychologische Physiologie*) has already pointed out one grave inconsistency of Meynert's in regard to the nucleus dentatus (seq.), and there are other impeachable points in the system. This alters nothing of the fact that Meynert is the pioneer of *applied* cerebral anatomy, and this may be a proper place to dispose of some objections made against his projection theory.

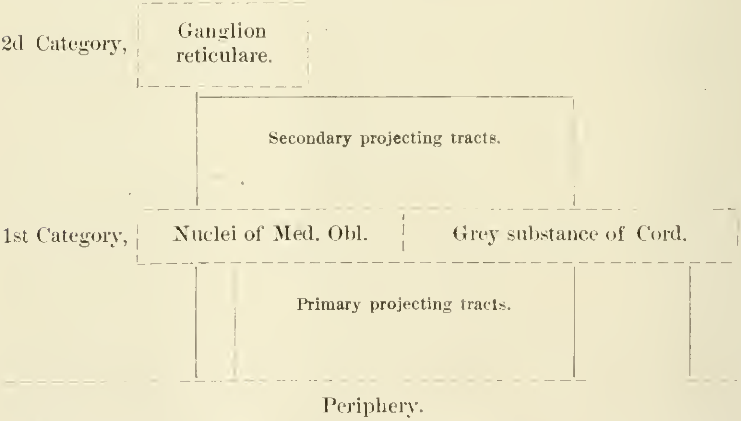
Thus Flechsig ventures to question the greater part of Meynert's deductions on the strength of the fœtal development of the medullary strands. While we owe several valuable facts to this author, yet his results as a whole show very little of the positive beyond what had been discovered on pathological and physiological or anatomical grounds, by Türck, Stilling, Schiefferdecker and Meynert. His method has been over-rated, and such results as his methods have enabled him to obtain in the medulla must yet pass in review before competent anatomical critics. Such grave errors as the confounding of the acoustic nerve root with the inner division of the lower cerebellar peduncle render the value of these results quite problematical.

Flechsig's dictum that the nerve fibres of a given system become provided with myelin *throughout at about the same time*, is an error, for the *processus e cerebello ad cerebrum* becomes white much later in the supra-tegmental than in the infra-tegmental portion. And as if to show beyond any possible doubt that Flechsig's theory stands on very weak grounds, Rohon (*Untersuchungen uber den Bau eines Microcephalen-Hirnes*) has shown that the spinal fibres and those of the motor decussation were fairly developed in a monstrosity which had not even a trace of the pyramids above the decussation, the entire cerebrum not being larger than a chestnut. Thus showing that fibres unquestionably normally connected with a given centre, are not always dependent in their embryonic development on that centre, and on the other hand demonstrating that a fasciculus does not develop in all its length progressively or simultaneously.

The objections of Forel are based on much safer grounds, and this writer, who if he errs at all, errs on the commendable side of excessive caution, has made several corrections of Meynert's theorem, which are now to be accepted as final (see relations of Corpora Quadrigemina, Corpus Geniculatum Externum, Corpus Luysii, Fasciculus longitudinales post. Tegmenti). They have, we hear, been accepted by Meynert himself, at least in part.

But in his valuable and exhaustive monograph on the tegmentum (*Archiv fuer Psychiatrie*, VII.) he makes certain demands upon the cerebral anatomist who would offer an anatomical basis for the physiological theory, which can never be realized. He insists that the fibres of a fasciculus must be traced to a ganglion with which it seems to be connected, before that ganglion and fasciculus can be ranged in a system. Now this is but rarely possible even with the nerve roots, and to insist on this point would simply be a bar to cerebral anatomy. No one has yet seen a single fibre of the optic nerves enter the cells of the optic lobes, and yet no one can doubt that they do so from the concurrent testimony of comparative anatomy and physiology! No one has ultimately traced the hypoglossal fibres in any one section to the hemispheres, and yet we know almost their exact course from pathological experience! There can be no doubt of the relation between the olivary body and the cerebellum, and yet no one has seen the fibres ever connect with the cells of that body! True, many of Meynert's deductions were purely hypothetical, and it would have been well if that author had not through his categorical style given the impression that he presented as facts what were merely ingenious deductions *founded* on facts. But he is not open to the charge which Forel makes (from a strange misunderstanding), that he claimed to trace the fibres of all the projection systems in longitudinal sections of the brain. He traced whole *fasciculi* thus, and was justified in so doing, not individual *fibres*. As no fibre runs the length of the brain without repeatedly changing its course, no single fibre can be traced any considerable distance. But a fasciculus can usually be safely traced, unless, like certain spinal bundles, it exhibits the *false appearance* of a compact bundle by gradually replacing fibres which it loses on one side by receiving fibres on the other.

§ 37. It follows from the preceding (§ 34, 36) that the medulla oblongata contains *both* the enumerated projecting systems, for we have the cranial nerves representing a first projection tract, their nuclei a first ganglionic category (the original central tubular grey), while the reticular ganglion represents the second ganglionic category. Now just as the second system is superimposed on the first system of the spinal cord, it is superimposed on the first system of the medulla oblongata, and this through fasciculi joining *some* of the cells of the reticular ganglion to the cranial nerve nuclei. These fibres represent the secondary projecting tracts. The projecting systems, so far as traced, present the following arrangement:



Wherever the cerebral anatomist encounters a large cell group, his first question is as to its connection with neighboring fasciculi. If these are known the function of the cell group can usually be deduced. The cells of the reticular formation are *known* to be connected with the nerve nuclei on the one hand, and with longitudinal fasciculi, which since they run into the cord, we infer to terminate either in the grey matter (or the nerve roots directly), for nerve fibres do not terminate with, as it were, blind ends. Now in the *mammalian* brain the reticular ganglion lies scattered among fibres which come from higher centres, and the interpellation might be made whether, after all, the reticular ganglion is not a mere intercalar station for fibres derived from a higher source. That *originally* the ganglion was an independent station, it can be answered, there is no reasonable doubt, for in reptiles this body of cells is too considerable to account for a termination in them of the *few* cerebral fibres possessed by these animals. And, on the other hand, the vertical strands are notably increased in their passage through the field of the medulla oblongata.

The medulla oblongata, with its reticular ganglion, seems to be the great rhythmical centre. In fish, the movements of the operculum and mouth; in sharks, those of the spiraculum; in perennibranchiate amphibians, the branchial tree; in the infant, the suctorial muscles; in all vertebrates, the movements of deglutition, of the heart and the respiratory muscles; *all* movements presenting a more or less regular *rhythm* are under the control of the medulla oblongata.

The early differentiation of this part of the cerebro-spinal axis is, without doubt, related to the early manifestation of rhythmical movements in the embryo, and their predominant importance in lower animals. Not that we intend to exclude the possibility of a rhythmic movement being purely spinal, nay, even controlled by *peripheral* ganglia (heart of embryo), but that a higher development implies the concentration of rhythmic innervations at some point where that anatomical association may be effected, which is the expression of the mutual influence which these movements exercise amongst themselves. (Seq.)

§ 38. The scattered ganglionic matter of the medulla oblongata, although not a part of the central tubular grey in the adult, is *derived* from it in the embryo. All through the primitive medullary canal we observe a tendency for the central tubular grey to send out peninsular processes, and to scatter its cells through the contiguous white substance. The peninsular processes are usually drawn out in the direction of the nerve roots to form the cornua in the cord, and the nerve nuclei in the medulla oblongata. The scattered cells in the cord become indifferent bodies and atrophy, or are utilized in the formation of the connective substances; but in the medulla oblongata they form accumulations crowded away from the nerve nuclei by white substance, and thus form the reticular ganglion.

Anterior to the proper field of the medulla oblongata, similar scattered cells form the basis of the intercalar ganglia—which we shall speak of later on—and further forward on the sides of the third ventricle they form a massive accumulation —the *thalamus*. We shall, for the better comprehension of the relations of this body, be again compelled to refer to the embryonic development of the brain.

§ 39. The first indication of the cerebral formation is a pyriform enlargement of the anterior end of the medullary tube. This enlargement is like the parent tube—hollow. The walls of the enlargement (*primitive cerebral vesicle*) are composed of the same elements, arranged in the same radiatory manner as the walls of the rest of the medullary tube. Through all subsequent morphological changes, the cellular elements of the brain hence maintain a vertical position to the surface contour of their respective locality, unless their position is disturbed by interrupting fibres.

§ 40. The primitive cerebral vesicle exhibits two constrictions, which subdivide it into three lesser segments, each of which contains a more or less round cavity, connected with the other cavities by the constricted lumina. These vesicles we designate as the (1) *fore-brain* (prosencephalon); (2) *mid-brain* (mesencephalon); and (3) *hind-brain* (prosthencephalon).

§ 41. The fore-brain undergoes a subdivision in the median line (median longitudinal fissure), which division does not extend all the way, however, but leaves the posterior part of the fore-brain untouched. This latter portion constitutes, according to anatomists, a separate division of the fore-brain, and is known as the *thalamus* (thalamencephalon). In the embryo, as well as in the adult, in the lowest, as well as in the highest animals, the thalamus maintains both a direct anatomical continuity and close physiological relation with the anterior segment of the prosencephalon, which, split in two by the posterior longitudinal fissure, we designate as the *cerebral hemispheres.*

From the fact, to be developed later on, that the thalamus and hemispheres constitute a structural continuity, and exhibit a mutual dependency of the closest kind, we refuse to accept the fundamental distinction made by comparative anatomists (untenable as it is even on comparative anatomical grounds), and range the thalamus as a ganglion of the hemispheres side by side with the corpus striatum.

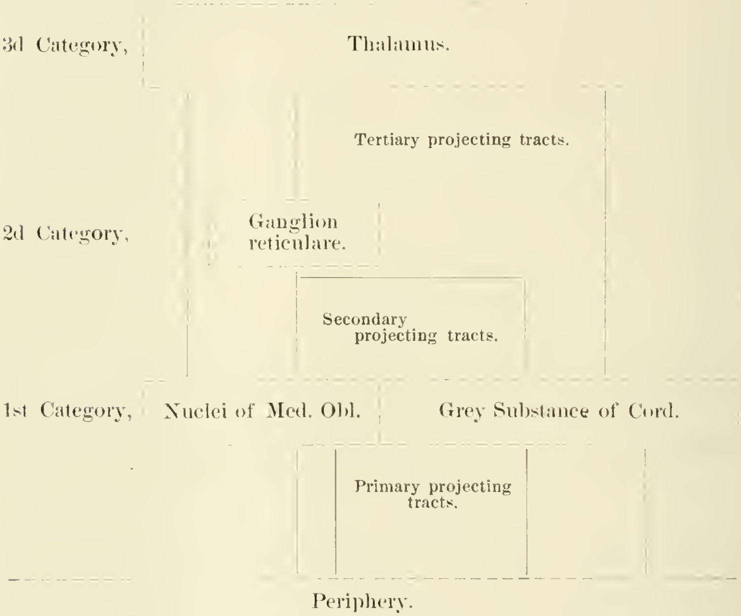
In the reptiles and amphibians this relation is manifested by the direct continuity of the cortical cells with those of the thalamus at the base of the fore-brain. In menobranchus (Fig. 5) the thalamus constitutes a hardly perceptible enlargement of the central tubular grey, continuous with the similar more external enlargement of the corpus striatum, and where this is absent, with the cortical grey directly. In the alligator a special ganglionic hypertrophy of the thalamus is induced by the termination in it of that part of the hemispheric inflection which corresponds to the cornu ammonis, a relation which is but slightly modified in the lower mammalia (Fig. 7).

This is also, perhaps, the best place to briefly refer to the embryonic segments of the cerebral vesicle. It is ordinarily taught that after a certain stage, in which only three vesicles, as mentioned in the text (§ 40), are present, that by further subdivision these form six secondary divisions (German authors make only five; but Huxley, in perfect consistency with a plan which is in itself defective, makes six). These are: 1, the *rhinencephalon* (olfactory lobes and bulbs); 2, the *prosencephalon proper* (hemispheres); 3, the *thalamencephalon*; 4, the *mesencephalon*; 5, the *metencephalon* (cerebellum); and 6, the *myelencephalon.* The first three are produced from the fore-brain, the last two from the hind-brain. We do not accept this classification, first, because it has proven the chief hinderance to homologizing the brains of the lowest vertebrates (as witness the Stieda, Michlucho-Maclay and Gegenbaûr controversy); then also because the corpus striatum should be raised to as distinct a position as the thalamus; and, finally, because the cerebellum originates as a local hypertrophy, *not a distinct segment* of the medulla oblongata. We hence admit only three cerebral vesicles.

§ 42. Although from our point of view the thalamus is in parallelism with the corpus striatum, in so far as they are both developed from the same cerebral sub-vesicle with, and are both dependencies of the cerebral hemispheres, there are certain differences. Just as the thalamus was situated between the balance of the prosencephalon and the mesencephalon, it shares characteristics of the hemispheric ganglia on the one hand, and the automatic ganglia on the other. It is physiologically as it is anatomically a connecting link between the cerebrum and the medulla oblongata.

In animals whose cerebral hemispheres are poorly developed, and where on account of the defective character of fibres which pass directly to the corpus striatum and cortex, the relations of those fibres which enter the thalamus from below are best studied, it can be seen that the strands which pass through and around the reticular ganglion enter the thalamus and even connect with its cells. Lesions of the thalamus are known to produce certain abnormalities of position, distortions of the bodily axis, and consequent disturbance of locomotion; for this we have the following anatomical basis:

The strands which enter the thalamus can be traced both from the lateral and posterior columns of the cord. More fibres enter it from below than connect it with the rest of the hemisphere (by emerging above), hence some of these strands must terminate in the thalamus without directly connecting with the hemispheres or corpus striatum. On the other hand anatomical research renders it highly probable that *some* of the strands connecting the cord and thalamus are interrupted by the cells of the reticular ganglion. We have thus an intricate arrangement of the ganglia and fibres which we represent in the annexed diagram.



The thalamus rises to the rank of a ganglionic category higher by one in the scale than the reticular ganglion. It is a representative of the *third category* and the thalamic fibres represent tertiary projecting tracts.

How the position of the thalamus as an independent centre is encroached on in the highest animals and how its relations are complicated in man, will be discussed in the limits of this chapter: the manner in which the fibres terminate and connect, in the chapters following. For the present we will content ourselves with pointing to the fact that when we speak of certain fasciculi being interrupted by cells of the reticular formation (reticular ganglion) we do not mean to say that the same fibres connected with thalamic cells can be identified as the ones connected with the cells in the reticular ganglion, but that the fibres of the *same fasciculus* are seen to be so connected. From this we draw the above inference. Meynert has named the thalamic fibres "motorisches Feld" of the human isthmus, and speaks of the cells of the reticular field (which we have named reticular ganglion in their aggregate) as scattered depots of the cranial nerve nuclei, agreeing in this respect with Deiters. True it is that on the one hand these cells do connect with the nerve nuclei, but we have offered another explanation for this relation (§ 37), one which does not fail as Deiters'-Meynert's explanation fails to cover that voluminous accumulation, known as "Kern des Seitenstrangs" (nucleus funiculus lateralis).

§ 43. Since the thalamic fibres, whether interrupted by the scattered cells of the isthmus or not, can be traced into both the lateral and posterior columns of the cord, it follows that the thalamic cells must be mediators between sensory impressions originating in the trunk and extremities and the movements which are determined in these parts. At the same time we remark that this ganglion in higher animals is not a mere homogeneous aggregation of nerve cells, but that these cells are arranged in definite groups (Fig. 7), which show considerable variation in the animal range and are separated by transmigrating fibres which enter, are lost in or connect with these different accumulations. This anatomical complexity seems to be the expression of a co-ordinating faculty. Anatomically it would be hazardous to infer such a faculty from the mere structure, but physiologically and pathologically the anatomical inference becomes entitled to consideration as at least a very reasonable theory (seq.)

§ 44. The portion of fore-brain anterior to the thalamus, undergoes a subdivision into the two cerebral hemispheres. These are in the urodelous amphibians of a remarkably simple structure. The cells are congregated around the ventricular cavities, just as those of the cord are in all animals in relation to the lining of the central canal. In man and higher animals the grey matter, with one local exception to be alluded to, is separated from the ventricle by the massive development of white substance, but in the embryo the white substance being absent, we have the same relation as in the urodela. Therefore the hemispheres are originally a part of the central tubular grey matter, and the same transitions between the epithelia lining the ventricle and the cells of the cortex can be traced, as were referred to in the case of the cord and medulla oblongata (§ 26).

In simpler forms, the entire thickness of the hemisphere is ganglionic substance (Fig. 5), in higher ones, white substance is developed, but not as in the cord on the outside of the ganglionic matter, but on its *inner aspect, the conducting fibrils separating the future cortical nerve cells from the epithelium* lining the ventricles (Fig. 6.) As we progress upward in the scale we observe that both the thickness of the white substance and especially of the grey substance undergo an enormous increase.

As the existence of a ganglionic depot without associating or projecting connections would be nothing less than an absurd assumption, it is to be concluded that in the animals possessing cortical without medullary substance, the conducting substance is fibrillar. This we have found actually to be the case, and the more searching inquiries of Schmidt ("Structure of Ganglionic bodies," *Journal of Nervous and Mental Disease*, 1879), have confirmed for the *amphiuma* what we have found in the *menobranchus*. That in the simplest forms, the ventricular epithelia may themselves be of a nervous nature we consider highly probable. Fibrillæ which are unquestionably nervous I have traced among and around the epithelial nuclei, in the true reptiles as well as in the amphibia. Schmidt (*loc. cit.*) has done the same, and Rohon (*Das Selachiergehirn*, Denkschriften der Kaiserlich. Akad., 77), remarks the great similarity of certain epithelia from the fourth ventricle to the epithelia of the peripheral sense organs. In this regard it deserves mention that one of the great masters of cerebral anatomy, Stilling, to his dying day, held all the epithelia surrounding the central canal of the cord to be of a nervous nature. While we should conclude from their relative atrophy in the human being that their nervous functions had become unimportant and perhaps entirely supplanted by secondarily developed and specialized ganglia, yet we would accord weight to the statement of Stilling as establishing a widespread homology of the nervous epithelia.

While there is a marked contrast between the hemispheres and cord (as stated in the text) with reference to the development of the white substance, it should be stated that certain convolutions, particularly those related to the olfactory lobe, develop white substance on the *outside* as well as on the inside of the grey matter. These gyri are the oldest genetically, and atrophy in man, showing that their functional importance is less in the latter. In the accompanying three figures a black space on the outer side of the cellular layer represents molecular substance belonging to the cortex proper; it is like all the molecular substances, relatively most massive in lower animals, and already the lower mammalia (Fig. 6) show less of it than the reptiles.

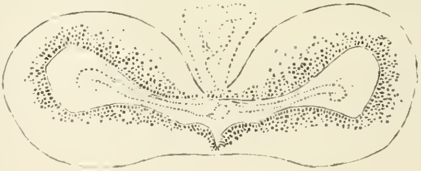


Fig. 5.

Transverse section of hemispheres, from American proteus (*menobranchus lateralis*). The dotted contour represents the tela choroidea, detaching the choroid plexus into the here common ventricular cavity through the atrophied prosencephalic roof or lamina terminalis.

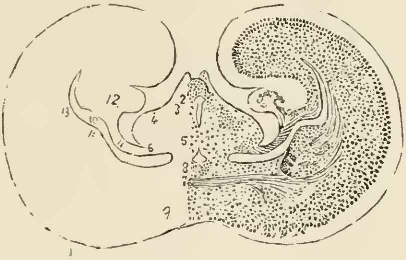


Fig. 6.

Same from small European bat (*vesperugo noctula?*): (x) barren layer of cortex; (12) cornu ammonis; (8) undifferentiated basilar grey; (9) cortex continuous with same; (13) white substance (corona radiata); (14) corpus striatum; (7) olfactory decussation (commissura ant.); (10) lateral ventricles; (12) cornu ammonis; (2, 3, 4, 5, 6) nuclei of thalamus; (6) for termination of fornix (11).

§ 45. We stated (§ 44) that with one local exception, all the grey matter of the hemispheres became separated from the ventricles and their lining by the developing white substance. The exception in question is at the floor of the ventricle, where the white fibres, swerving from their course, cut into the grey substance in such a way as to leave one portion in contact with the ventricular lining, *the corpus striatum*, driving the other part out, as they did the rest of the cortex (Fig. 6).

Before this differentiation occurred, the anterior portion of the hemispheres developed a distinct appendage, containing a prolongation of the lateral ventricles, and termed, from its relations to the *regio olfactoria*, the *olfactory lobe.* This olfactory lobe and the corpus striatum are in direct continuity, and manifest intimate relations throughout the animal range.

Evidently the sense of smell is one of the most important in lower animals. However much this sense may have lost in importance in the human species, which makes up for the deficiency in countless other ways, and in birds, whose acuteness of vision vicarates for the almost complete absence of this sense, yet in all water breathers it is one of the conditions of their existence. Even in land animals, not only in the carnivorous, but also in the herbivorous species a great portion of the psychical life is based on olfactory conceptions.

Now water breathers constituted the ancestry of the vertebrate sub-kingdom, and it is reasonable to suppose that the olfactory lobe constitutes one of the most ancient differentiated segments of the central nervous system. In the myxinoid fishes and the lamprey *the* CEREBRAL HEMISPHERES THEMSELVES *are mere appendages of the olfactory lobes, and hardly half their size.*

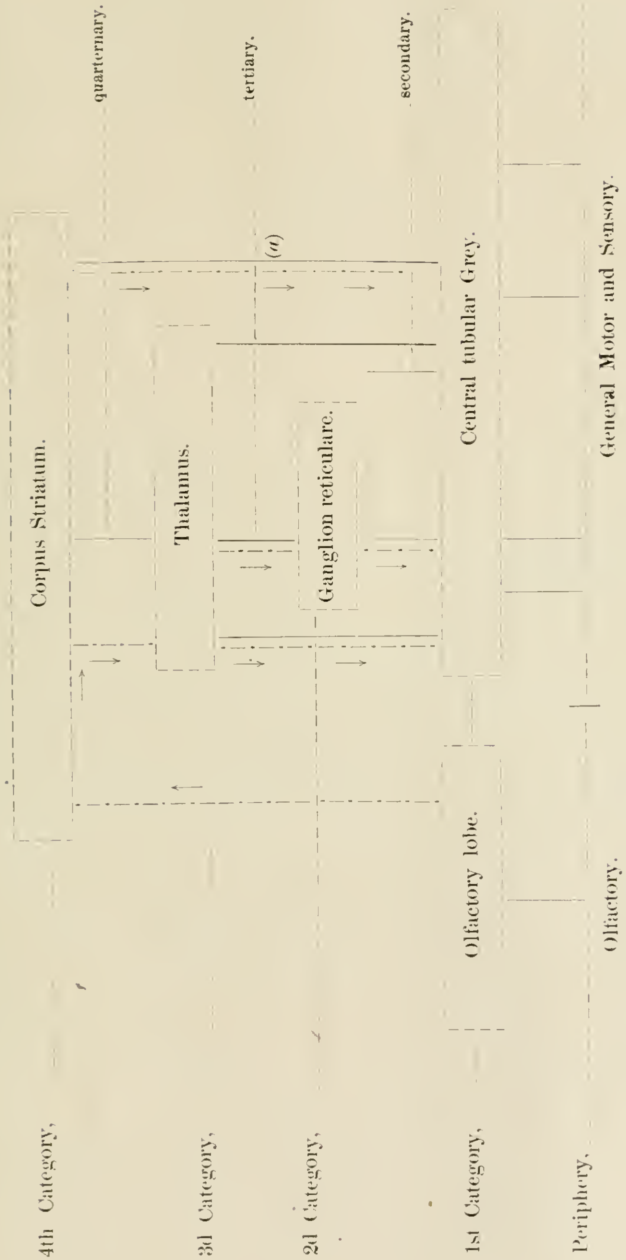
This fundamental importance of the olfactory lobe implied a corresponding development of the associating conductors, which bring the impressions of this sense into such relations with *the motor* apparatus, as to permit of their utilization for the purposes of the animal economy.

These conductors could travel but in one way, namely, through the corpus striatum and the thalamus or sub-thalamic region, after passing the corpus striatum. This is in lower animals; in higher, more complicated relations are established.

Now the olfactory lobe clearly represents a first category of grey matter, for its periphery is projected in it directly by the filamenta olfactoria without the intervention of any intermediate ganglia.

But the corpus striatum, superior to the thalamus in position, and receiving fibres which have passed through the latter, is equivalent to a fourth category of grey matter, as we will establish more fully.

Since the fibres of the secondary projection from the olfactory lobe connect with the corpus striatum, we have a secondary projecting tract terminating in a ganglion of the fourth category. This is one feature which gives the olfactory nerve a high position in the scale of the cranial nerves—one, in fact, which establishes a profound difference between it and other cranial or spinal nerves, except one—the optic—which we shall find to merit an equally special position.


4th Category,
quarternary.
Corpus Striatum.
3d Category,
tertiary.
(a)
Thalamus.
2d Category,
Ganglion reticulare.
secondary.
1st Category,
Central tubular Grey.
Olfactory lobe.
Periphery,
General Motor and Sensory.
Olfactory.

In this diagram the broken and dotted line with the arrow represents the path taken by a sensory impression, traveling to the olfactory lobes, thence transmitted to the motor ganglion, and down through the peduncular axis to the motor periphery. The multiplicity of paths emerging from the corpus striatum is introduced to represent the *anatomically* various *possible* tracks which any motor impulse may travel. Of their conditions of development more anon; but in passing, we may indicate that the very existence of both direct and more or less interrupted fasciculi connected with the same category of grey matter, shows that a *strict classification of the projecting fibres into primary, secondary, tertiary, etc., cannot be practically carried out.* We employ such a classification merely as a provisional and theoretical one, in order to systematize somewhat the intricate tracts which connect the different ganglia with the more or less remote periphery, and to furnish a basis for a subsequent physiological speculation. The same applies to the ganglionic "categories."

§ 46. Not quite as early in its appearance, and not as pronounced in physiological importance (in the lowest vertebrates) we have the visual periphery developed. In the higher vertebrates so prominent does this sense become, and so intimate its relations to the psychical life, that, *ab initio*, the embryonic trace of the retina is developed in *direct continuity with the nervous axis.* Originally connected with the central tubular grey matter of the third ventricle, it secondarily develops a projection in the *dorsal part of the second* cerebral vesicle—the mesencephalon. Its primary projection loses in importance and becomes an atrophic remnant in man, probably as devoid of function as it is devoid of any special structure and connections. This is Meynert's *basal optic ganglion.*

§ 47. In entering the mesencephalon, the optic nerve spreads out on the surface of this vesicle. At the same time there is developed within the ganglion other conducting substance, which separates the outer cells from the inner.

The inner cells retain the position and the signification of a central tubular grey; the outer cells constitute the *essential ganglion of the optic lobes*, and exhibit a *cortical* structure. This fact is of some significance. The cortical structure is one which is employed wherever a given periphery is to be accurately and exhaustively projected. It is manifested in the olfactory and optic lobes in the cerebrum and cerebellum.

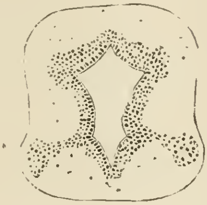


Fig. 7. Optic lobe of menobranchus.

§48. Thus, while the optic lobes of the lowest animals (Fig. 8) show merely the central tubular grey, in higher ones we find, 1st, the superficial white substance constituting a primary projection path, and derived from the optic tract; 2d, a grey ganglionic layer, the *primary centre of vision*;* 3d, a deep, white layer; and 4th, the central tubular grey.

The deep white matter connects the visual centre with the oculo-motor nuclei in the first place, and with undetermined nuclei of the spinal cord in the second. As the fasciculus which establishes this latter association gradually becomes exhausted in the cervical region, and maintains its anatomical continuity as a fasciculus only by receiving fresh accessions of fibres from other sources, we may assume it to terminate in the nuclei which control the reflex movements of the head and neck.

The optic nerves and tracts constitute a primary projection tract; the cortical grey of the optic lobes a first ganglionic category; the deep white substance is an associating tract of the lowest or first order, connecting two ganglionic stations of the first category.

Omitting the already assumed projecting systems, we may represent the above relations in the subjoined plan:

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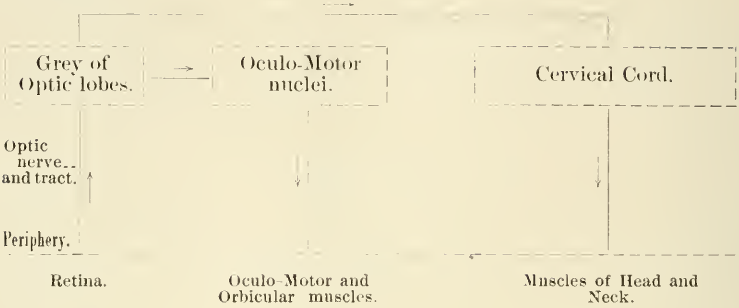
*In a physiological, but not in a phylogenetic sense.

1st Category.



Although we have here ranged the optic lobes in the first category, it is seen from the above diagram that they extend their sway over a considerable motor territory, and like the olfactory lobes occupy a special position, in contrast with the other cranial nerves.

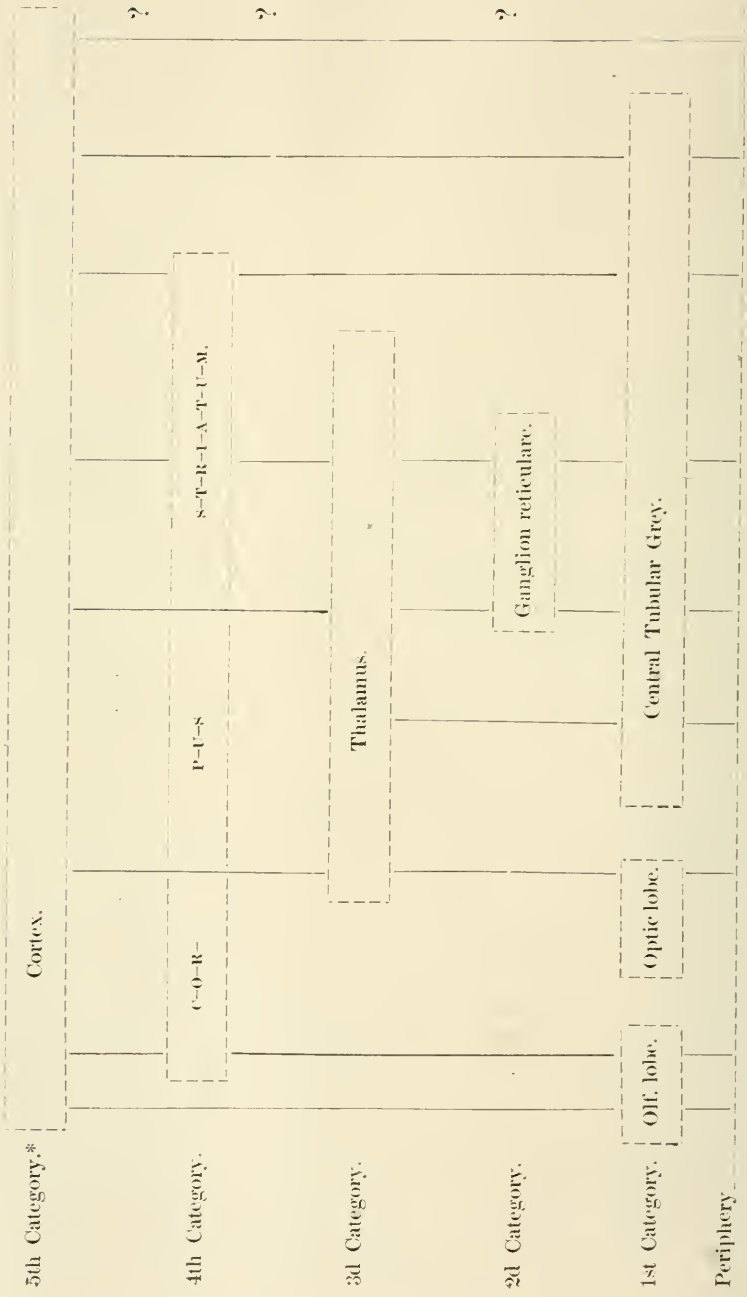
§ 49. We have now traced (theoretically for the present) the anatomical basis of certain co-ordinations; co-ordinations which were based on associations between the sensory and motor peripheries, and which according as they involved a lower or higher category of grey matter were more or less limited in their range of complexity. The first projection systems simulated so many simple mirrors, each of which reflected a portion of the object presented to the series, while the second and higher systems simulated larger mirrors which collected by re-reflection the sum of the images projected on the smaller mirrors, and combined them into pictorial unities. But as yet there were still several unities: the optic lobes representing one; the olfactory and corpus striatum another; the thalamus and its dependencies a third. To unite them in function, mutual associations became necessary, and these thenceforth join in anatomical embrace all the great basi-cerebral ganglia. This *association is the basis of the highest so-called automatisms.*

§ 50. It is almost a rule in morphology that where several structures co-exist in functional union, one or the other will preponderate, and the higher the structural union becomes developed, the more decidedly does the superiority of the predominating member become marked.

The olfactory lobe bore such important relations to the life-history of early vertebrates, that we are not surprised to find the *cerebral hemispheres* developing at first as mere appendages of the olfactory lobes (§ 45).

The corpus striatum representing a fourth category of the projection grey, projected in turn its innervations in the hemispheric grey, thus constituting the latter a *fifth order.* Then the thalamus found besides the indirect projection through the corpus striatum already referred to (§ 45) a direct connection with the cortex. The optic lobes followed, first by indirect projection through the thalamus, then by a special circuit, through the ganglion geniculatum externum (seq.).

With the development of these highest projection fibres the cerebral hemispheres gradually encroached on the independency of the lower ganglia, until in its maximal development as found in man, it resembles a great empire which holds a number of tributary states in sway under a common powerful rule. The anatomical unity now attained, finds its parallel psychical culmination in that more perfect consciousness of the *ego* which is peculiar to man.



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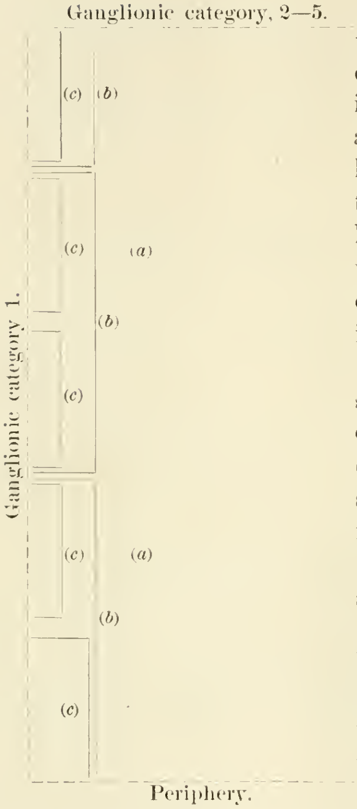
*It will be hardly necessary to remark that topographical representations are not even remotely aimed at in these diagrams.

§ 51. In this preliminary sketch, it being purely our object to formulate the principles of the cerebral architecture, we shall not find it necessary to discuss the relations of the cerebellum, or of the intercalar ganglia. We would, be merely repeating the same theorems and deducing the same laws as those we are now preparing to make a summary of.

All through these last paragraphs we have found one striking feature in the elaboration of the projecting tracts, namely that, in higher developments, the fasciculi show a tendency to emancipate themselves from the interruptions offered by intermediate ganglionic categories, in short that the tendency is, to establish a direct communication between the cortex and the central tubular grey matter. It is the same tendency which led to the development of the longitudinal tracts of the cord, thus establishing a readier association than the circuitous route furnished by the fibrillary net-work of the grey substance.

The course of this development is determined by two physiological laws: the first is, that, the greater the distance traversed, the longer will the impression take to travel to its destination; the second is, that every ganglionic element to be traversed delays the transmission of the nerve current.

In the diagram, the conducting path (*a*) is far more perfect as a mechanism than (*b*), and (*b*) is in its turn more perfect than (*c*).



§ 52. While the first law if it were the only one which governed the conducting mechanism, would tend to produce an atrophy of the intercalar ganglia, the second, which up to a certain point has the same tendency, has a conservative element in it, which operates against such an entire abolition of the interrupting stations.

It is desirable for certain physiological purposes, that the mechanism by which nerve currents can be arrested at certain points, should be maintained. Many inhibitions doubtless take place by virtue of this interrupting arrangement.

§ 53. In order that all physiological necessities may be complied with, we must assume that in man, the projecting system falls into two groups: the one which shows a tendency to emancipate itself entirely from the interrupting ganglia, the other which maintains its connections with the latter. The continuous tract of the internal capsule, pes pedunculi, vertical pons fibres, anterior pyramids, and part of the lateral columns of the cord, is an example of the most direct variety. The posterior part of the corona radiata, the thalamus, thalamic fibres, reticular ganglion, other portions of the lateral columns, represent a form of the indirect variety. Both are necessary to the purposes of the organism, but the indirect variety being devoted to the organic needs, and these needs being the same in all animals, or nearly so, shows less variation in the zoological range than the direct. The latter which typifies the domination of the intellectual centres rises in a mathematical ratio as we approach the human being. So that in man, while the *automatic ganglia and tracts remain about the same in mass as in the other mammalia, the intellectual ganglia rise far above them, and this gives rise to the apparent but not real diminution of the automatic systems observed in the human brain.*

§ 54. Certain actions which, while the hemispheres and higher ganglia were yet poorly developed, were presided over by the central tubular grey, or its immediate derivatives, become translated *bodily* to higher systems in higher animals. This accounts for the atrophy of finally useless centres which persist only in accord with the uniform plan of development manifested in any group of animals. Thus the optic lobes, though extremely rudimentary in blind genera, still are present. The basal optic ganglion (§ 46) is another example of the persistence of the supplanted and physiologically defunct centres; the grey of the locus perforatus, and the interpeduncular ganglion hold similar positions.

Thus far we have traced cursorily, because for merely provisional purposes, the general plan of the cerebral architecture. We have seen that the entire central nervous axis was a development from the central tubular grey, a portion of which retained its primitive position in the shape of the first ganglionic category.

This *axial* grey matter is genetically the earliest, anatomically the easiest studied, and physiologically the simplest of all the ganglionic systems; and just as structurally it was the primordial fundament from which all the higher ganglia became gradually elaborated, so functionally it is the basis on which they rest.

Our description of the Architecture and Mechanism of the Human Brain will, therefore, begin with the discussion of the axial ganglia, more familiarly known as the cranial nerve nuclei proper, and of their connections.

(*To be continued.*)

Art. V.—A NEW FUNCTION OF THE OPTIC THALAMI.

By Isaac Ott, M. D.,
Late Lecturer on Experimental Physiology, University of Pennsylvania,
and G. B. Wood Field, Stud. Med.

THE influence of the nervous system on the peristalsis of the intestinal canal is one of the most intricate questions in experimental physiology. That nerve centres have an influence on the intestinal movements has been shown by the researches of Budge and Valentin, who state that irritation of the optic thalami, corpora striata and the tubercula quadrigemina excite the peristaltic movements. Schiff has seen movements of the intestine ensue after irritation of the medulla oblongata, cerebral and cerebellar peduncles and pons varolii. Longet* obtained results so variable that he was not able to come to any conclusion.

Ludwig and Haffter from experiments on rabbits arrived at the conclusion, that the splanchnics had neither an inhibitory nor an exciting effect on the intestinal movements, and after section of the splanchnics there ensued neither increased peristalsis nor diarrhœa.

Pflüger † first observed after irritation of the splanchnics that the small intestines became quiet or rather were inhibited by a direct action on ganglia seated in the walls of the intestine.

Nasse ‡ found the splanchnics not only to contain inhibitory but also exciting fibres, and that irritation of the dorsal segment of the cord arrested the intestinal movements.

Ludwig and Kupffer arrived at the same conclusion as did Nasse.

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* Longet, *Traite de Physiologie*, tome premier, Paris, 1868.

† *Ueber das Hemmungs-Nerven System für die peristaltischen Bewegungen der Gedärme*, Berlin, 1857.

‡ *Beiträge zur Physiologie der Darmbewegung*, Leipzig, 1866.

That there is an inhibition by the central nervous system on the intestinal movements is proved by the researches of Goltz,* when he removed the medulla in frogs and found the peristalsis much more active.

Wilson Philip † observed a long time back that the movements of the small intestines continue a long time after removal of the cerebro-spinal axis.

I‡ have already shown in another place that in the optic thalami are seated centres inhibiting the rhythmic movements of the sphincters of the rectum and vagina. It subsequently occurred to me to determine if any centres existed inhibiting the intestinal movements. That centres exciting intestinal movements exist has been proven by other observers whose results I can also confirm.

Method :—The animals employed were cats and rabbits, which were fastened on Czermak's holder and etherized. Then two holes were trephined into the skull for the entrance of the electrodes. The location of these trephined openings was made directly over the optic thalami. The electrodes consisted of needles insulated to near their points by sealing wax. The induction current sent through them was derived from the secondary coil of a DuBois apparatus. The cell driving the apparatus was a small zinc and carbon one. The strength of the current was such as to be strongly felt by the tongue. Then in rabbits under ether the abdomen was opened and the normal rate of peristalsis of the abdomen and the remaining part of the small intestines watched. The temperature was about 85° F. After the normal rapidity of the intestinal movements was determined, then the electric current was turned on, when the small intestines came to rest in diastole.

This arrest of persistalsis was not due to vaso-motor irritation, as the electric excitation was made some distance above the seat of the monarchical vaso-motor centre; and if this centre was irritated, then anæmia of the intestine would be produced, which state, according to Nasse, produces lively peristalsis. During the irritation the blood-vessels of the intestine did not become pale, but, on the contrary, became hyperæmic, by the exposure to the air. It has been stated by Van Braam Houckgeest that anæmia arrests peristalsis, but this is contradicted by the experiments of Sanders Ezn, who affirms that the amount of blood in the vessels has no influence whatever on the peristalsis.

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* *Pflüger's Archiv*, 1872, p. 616.

† Longet's *Nerven-System.* Erster Band, Leipzig, 1847.

‡ *Journal of Physiology*, vol. 11, No. 1.

That these centres of inhibition were not seated in front of the optic thalami, was proven by experiments where the needles were inserted in front of the thalami, and no arrest of the peristalsis took place. That these inhibitory centres are not seated behind the optic thalami, is rendered probable, as only weak currents were used, and when the needles were pushed backward, no arrest of intestinal movement took place. Further, in cats, after section behind the thalami, the peristaltic movements of the duodenum became quite active. The method of operating on cats was as follows: The animals were bound down on Czermak's holder, etherized, a trephine placed on each side of the skull behind and above the mastoid processes, and an opening made in the skull. Then the abdomen was opened, and a section of the brain made behind the thalami with a spear-shaped knife. After the section, the usually quiet peristaltic movements became much more active.

The intestinal movements may be compared to those of the heart. Like the heart, the intestine can move independent of the central nervous system, from the ganglia of Meissner and Auerbach. Simple excision of a length of the intestinal canal proves this fact. Like the heart, it is under the excitation of nerves running in the splanchnics and pneumogastrics; like the heart, the peristalsis is under the inhibitory control of nerve fibres running in the splanchnics, having their remote inhibitory connection in the thalami optici; like the heart, the movement of the intestine is under the control of drugs, which paralyze the inhibition of the splanchnics, as takes place when atropin and nicotin are given; like the heart, the peristalsis is influenced by emotions, for fright causes a movement of the bowels by a paralysis of the intestino-inhibitory mechanism, thus allowing the exciters of peristalsis full control.

Appended are some of the experiments upon which the preceding statements are based:

Exp. 1.—Rabbit. Etherized; two holes trephined into the skull; abdomen opened, and the peristalsis found to be quite active; electrodes thrust through the trephined opening into optic thalami, when a weak induction current was turned on, the peristalsis of the duodenum and small intestines was arrested in diastole. One experiment was repeated several times with the same result.

Exp. 2.—Rabbit. Etherized; trephined; bowels exposed; needles thrust into the optic thalami, when the electric current arrested the peristalsis.

Exp. 3.—Large rabbit. Etherized; trephined; abdominal contents laid bare; electrodes pushed into the thalami, when the duodenum was arrested in diastole.

Exp. 4.—Small rabbit. Etherized; trephined; bowels laid bare; electrodes thrust into the optic thalami, when the peristalsis of the small intestines was arrested.

Exp. 5.—Small rabbit. Etherized; trephined; intestines exposed; electrodes thrust into the optic thalami, when the peristaltic movements were arrested.

Exp. 6.—Small cat. Chloroformed; trephined near the mastoid process; abdomen opened, and the peristalsis of the duodenum watched. A section was made behind the optic thalami, when the duodenum exhibited lively movements, which soon came to rest.

Exp. 7.—Medium-sized cat. Chloroformed; trephined behind and above the mastoid process; bowels laid bare by section of the abdominal muscles; section behind the optic thalami, when the peristaltic movements of the duodenum became increased, the adjacent portions of the small intestine being also more active.

Art. VI.—CEREBRAL TOPOGRAPHY.

By S. V. Clevenger, M. D.

LOCALIZATION in cerebral physiology and pathology, depending upon the composite nature of the brain, requires minute study of the shape and plan upon which this association or confederation of organs is arranged. Anatomy is indebted to Gratiolet and Leuret, not only for their investigations and accurate delineations of constant encephalic features, but for having reconciled and systematized the observations of their predecessors since the days of Aristotle.

An excellent article, by Pozzi, in the *Dictionnaire Encyclopedique des Sciences Médicales* (Paris), entitled "Circonvolutions Cérébrales," mentions the surprising want of uniformity among the older writers on anatomy, concerning the relative positions of these tortuosities of brain structure, and calls attention to a "figure of Spigel, *De corporis humani fabricâ*, lib. X., tab. 2." Pozzi ascribes this defect in anatomical books to the gross analogy, used by the authors, in likening the cerebral gyri to the *valvulæ conniventes* of the intestines.

This ignorance is not confined to the ancients, for on page 586 of Gray's *Anatomy* there occurs this passage: "There is no accurate resemblance between the convolutions in different brains, nor are they symmetrical on the two sides of the same brain." In a strict sense such a remark may be correct, but conveys an erroneous notion.

"Such authors," says the French encyclopedist, "are devoted to advancing the idea of the absolute variability in the disposition of the convolutions, the relative variability sufficing for a long time to justify prejudice against closer study."

The oldest works, of a precise character, on the cerebral anatomy of the primates (man and the ape) appear to be: Willis, "*Cerebri Anatome cui accessit nervorum, descriptio et usus*," London, 1664; Malpighi, "*De cerebro, et de cerebri cortice, opera*," London, 1686; Tyson, "*Orang-outang, sive*

Homo-sylvestris, or the anatomy of a Pygmie compared with that of a Monkey, an Ape, and a Man," London, 1699.

In the eighteenth century appeared *Cerebral Essays and Treatises* by Vieussens, 1716; Ridley, 1725; Albinus, 1725, 9; Morgagni, 1741; Zinn, 1749; Tarin, 1750; Lorry, 1760; Malacarne, 1780; Vicq d' Azyr, 1781, 83, 86, 90; Soemmerring, 1788, 92, 96, 99; Rudolphi, 1796. At this period the more evident convolutions of simpler form were described by Vicq d' Azyr and Soemmerring, the lobulus fornicatus and the external parietal convolutions. In this present century followed works by Chaussier, 1807; Gall and Spurzheim, 1808, 9, 10; Doellinger, 1814; Gordon, 1815, 17; Tiedeman, 1816, 21, 36; Lauth, 1819; Burdach, 1819, 26; Reil, 1822, 23; Serres, 1824; Laurencet, 1826; Rolando, who in 1828, 31, in his "*Della struttura degli emisferi cerebrali. In memorie della Reale Accademia della sciege di Torino,*" t. XXXV., p. 103, properly insisted upon certain fissures, and is said to have given a very exact description of the insula and "*processo anteroïdo cristato,*" or lobulus fornicatus. Hertwig, in 1826, published "*Experimenta quædam du effectibus læsionem impartibus encephali,*" anticipating the matter of localization in cerebral pathology.

Todd in 1847, wrote that Mayo (1827) had published "the best plates of the brain extant." Up to the time of Todd and Bowman's *Cyclopedia* the standard works on anatomy treating the subject, were those of Hildebrant, by Weber; Cruveilhier; Soemmerring, by Valentin; Meckel; Bell; Quain and Sharpey.

After Mayo, we have: Langenbeck, 1830; Arnold, 1838; Bourgery, Ribes, Leuret, 1839; Wilbrand, Baillarger, 1840; Van der Kolk, 1841; Parchappe, 1842; Foville, 1844; Todd, 1845; Burmeister, Stilling, 1846; Retzius, Solly, 1847; Ecker, 1851; Hirschfeld and Léveille, 1853; Huschke, 1854; Gratiolet, 1855, by whom a new era in encephalology was inaugurated; Krigma, 1855; Owen, 1857; Arnold, 1858; Ecker, 1859; Reichert, 1859, 61; Campen, 1859; Wagner, 1860; Huxley, 1861; Broca, 1861, 70, 71; Flower, 1862, 63, 64; Van der Kolk and Vrolik, 1862; Rolleston, 1862, 63; Wagner, 1862; Macleod, 1864; Barkon, Bradley, Lankester, Vogt, Turner, 1865; Pansch, Quain, Turner, 1866; Meynert, 1867; Bischoff, Ecker, Pansch, 1868; Ecker, Alix, 1869; Bateman, Broadbent, Gromier, Calori, Jensen, 1870; Nitsche, Sappy, Lussana and Lemoigne, Henle, 1871; Gromier, Bischoff, Gervais, Tuke and Fraser, Major, Hitzig, Mierzejewski, Schüle, Wernher, 1872; Turner, Mivart, Hugnenin, Ferrier, 1873; Gromier, Darwin, Pozzi, Maliniverni, 1874; Charcot, 1875; Richet, 1878; Pansch, 1879; not including those writing upon comparative anatomy, prominent among whom were Serres, 1824; Desmoulins, 1825; Leuret, 1839; Rymer-Jones, 1841; Carus, 1853; Owen, 1857; Luys, 1865; Stieda, 1870; Huxley, 1871; Meynert, 1872; Ferrier, 1873.

Pozzi, p. 340, *op. cit.*, says:

"Nous devons à l'anatomie comparée d'avoir débrouillé le chaos apparent des circonvolutions chez l'homme. Les premiers pas avaient été faits dans cette voie dès la plus haute antiquité. Erasistrate écrivait: Les circonvolutions sont plus nombreuses dans l'homme que dans les animaux, parce que l'homme l'emporte sur les animaux par l'esprit et par le raisonnement. A quoi Galien répondait: Je ne partage pas cet avis, car d'après cette règle, les ânes étant des animaux abruptis et stupides, ils devraient avoir un cerveau tout à fait simple, sans aucun sillon ou sinuosité, tandis qu'ils ont beaucoup de circonvolutions." (*De usu partium*, lib. 8, chap. 13.)

"Mais il a fallu de longs siècles pour que l'idée d'Erasistrate servît de guide à l'anatomie humaine."

This paper has for its object the suggestion of a rational scientific nomenclature for the exterior cortical portion of the brain. The necessity for this becomes apparent when examining the literature of the subject, rapidly assuming voluminous proportions as its importance grows evident to students.

The synonyms given display at once the difficulties encountered in applying the topographical terminology of one author to the text of another. Ecker, Turner and Huxley have done much to simplify and systematize the names of the convolutions and fissures, but the recent monograph of Pansch, of Kiel, entitled "*Furchen und Wülste am Grosshirns des Menschen*" (1879), indicates a system which in most respects is very desirable, and which in the main is followed in this essay. Wherever a departure is made from his scheme the warrant for it is found in the accepted usages of other investigators. In beginning this compilation I greatly underestimated the amount of labor it would involve; but if it proves useful in tending toward improvement upon the systems heretofore prevalent, I shall feel fully justified in having undertaken it.

THE PLAN.

Owing to the difficulty of properly classifying the convolutions into groups or lobes, such divisions can be of a general character only; and instead of laboriously manufacturing limits for lobes, the more rational method is the natural one of *Surfaces.* We have then the *External* or convex; the *Median* or plane, and the *Basilar* or inferior *Surfaces* of the brain. An arbitrary subdivision into lobes may now be made, for convenience in general reference, separating the external and median surfaces into *Frontal, Parietal, Occipital* and *Temporal Lobes*; the sulcus Rolando between the frontal and parietal and the fissura Sylvii bounding the temporal lobe, superiorly. The basilar surface can be divided in the same indefinite manner into *Occipital*, *Temporal*, and *Frontal Lobes.*

Pozzi objects strongly to the use of either lobes or lobules in the nomenclature, but as objections can be urged against any selection of terms for portions of the cerebrum, let us select the most convenient and proceed. Probably a better understanding can be reached by defining the terms mentioned above with those to follow:

Surfaces: Natural divisions of the cerebrum into External, Median, and Basilar.

Lobes: Arbitrary divisions of the surfaces into Frontal, Parietal, Temporal, and Occipital.

Lobules: Portions included between fissures or sulci, heretofore styled convolutions, gyri, meandri, processi, plis, circonvolutions, volutions, lobes and lobules.

Gyri: Indivisible or lesser divisions of the lobules, possessing noteworthy relations, shape, or character, bearing something of the relationship to lobules the atom does to the molecule, at present having no designation apart from lobules, convolutions, etc.

Fissuræ: Primary, somewhat deep, constant clefts or furrows, associating man with animals of inferior intelligence. Sulci has been used interchangeably with fissuræ, to designate the same furrows before Pansch's late work.

Sulci: Lesser cortical fissures or furrows constant in the adult human brain.

Sulculi: Inconstant or little sulci, or furrows having no regular location or relationship to the convolutions.

Rami: Constant branches of fissuræ or sulci.

Extremitates: Useful in designating parts otherwise illy defined, because the sulci break up into forks and sulculi on reaching the extremes of the hemispheres. Prof. Pansch uses three divisions: *Extremitas orbitalis; Extremitas temporalis; Extremitas occipitalis.* The latter is the least useful, owing to the clustering of gyri fully occupying that region. It is not possible to exactly define these extremities, but they may be considered as including parts dubiously divided by the terminations of sulci.

FISSURÆ.

1. FISSURA LONGITUDINALIS.

Synonyms: f. or scissura cerebri longitudinalis; superior longitudinal sinus; f. c. l. superior, inferior et horizontalis posterior; interlobular f.; f. magna cerebri; interhemispheric f.

2. FISSURA SYLVII. (Cum ramus ascendens [et ramus anterior horizontalis, Pansch]).

PANSCH: f. sylvii mit ram. anterior horizontalis und ram. ant. ascendens.

ECKER: f. s., mit ram. ascendens s. anterior und ram. posterior s. horizontalis.

BISCHOFF: Stamm vorderer senkrechter Ast und hinterer horizontal aufsteigendes Ast der sylvischen spalte.

HENLE: f. lateralis posterior et anterior.

BROCA: scissure de s., branche antérieure et branche ascendente.

TURNER: f. of Sylvius w. horiz. and asc. limb.

GRATIOLET: fossa de Sylvius.

POZZI: scissure de Sylvius.

LUSSANA and LEMOIGNE: scissura di Silvio.

RICHET: scissure de Sylvius.

VARIOUS: fissure or fossa sylvii, fissura anterior; f. ant., inferior et externa; f. inferior; f. transversæ.

Entitling this fissure a fossa is in favor with some, and for this there is some reason, especially when its borders are separated so as to bring into view the Insula. But the word *fissure*, implying nothing as to depth, and designating this great seam better *in situ*, seems more appropriate. Ecker and Pansch differ concerning what parts should be called rami, while Turner attaches the ramus ascendens to the præcentral sulcus, in which he is not imitated by other authors.

3. FISSURA OCCIPITALIS.

PANSCH : fissura occipitalis.
ECKER : f. parieto-occipitalis pars medialis et lateralis.
BISCHOFF : f. occipitalis perpendicularis interna.
HENLE : f. occipitalis perpendicularis.
BROCA : scissure occipitale.
TURNER : parieto-occipital fissure, interna et externa.
HUXLEY and CHARCOT : the same as Turner.
GRATIOLET : scissure occipitale int. et ext.-occipito-parietale.
L. e LEMOIGNE : scissura perpendicolare.
FERRIER : parieto-occipital fissure.
FOVILLE : scissure posteriore.

There would be more warrant for attaching the occipital to the sulcus parietalis than the parietal to the fissura occipitalis, for it terminates in the human brain before fairly becoming parietal. The extra lateral development of this fissure in the apes is sufficiently posterior to justify retaining the word occipital, wherever occurring.

O te te

4. FISSURA CALCARINA (cum rami ascendens et descendens.)

PANSCH, ECKER, FERRIER, TURNER, HUXLEY : fissura calcarina.
BISCHOFF : f. hippocampi.
HENLE : f. occipitalis horizontalis.
BROCA : scissure calcarine.
FOWLER'S CHARCOT : calcarine fissure.
L. e LEMOIGNE : scissura occipito-parietale interna. /S
GRATIOLET : scissure interne, s. des hippocampes.
RICHET : scissure en pied de cheval, branche superieure et branche inferieure de la meme.

This important fissure has been, by some of the older writers, shortened by conferring its anterior portion upon the fissura occipitalis; it has been amplified by others, who have included the occipital with it; and omitted still by others, who consider it as a ramus of the occipital.

The name *calcarine*, from its marking the position internally of the calcar avis or hippocampus minor, would be preferable to "interne" of Gratiolet, as too indefinite, or "hippocampi" of Bischoff, as the larger hippocampus might mislead in locating it.

5. FISSURA HIPPOCAMPI.

PANSCH, ECKER, FOWLER'S CHARCOT, HUXLEY : fissura hippocampi.
BROCA : rainure du grande hippocampi.
TURNER : dentate fissure.
RICHET, FERRIER : sulcus hippocampi.

The dentate of Turner, referring to the fascia dentata, seems to other investigators to have less claim than hippocampus major, after which they have named the fissure.

These five fissures constitute the primary constant and deep separations of the lobules of the human brain, and they link bimana to quadrumana, across the artificial division of Owen, which placed man as an order of mammalia in the sub-class archencephala; the ape standing at the head of the gyrencephala.

Classification by zoological persistence would require the introduction of rudimentary or assumed fissures possessing a remarkable constancy in types lower than man. Lussana and Lemoigne have attempted this in locating a small "solco inflesso" anterior to the sulcus Rolando to mark the carnivoral crucial sulcus. The olfactory and calloso marginal sulci would merit distinction as fissures in many instances on the typical plan. We are, however, to seek the convolutions of the human brain, and, conforming to Pansch, the cortical fissures become sulci.

Among primary or constant typical furrows of the cortex the first sulcus is the sixth in order.

6. SULCUS ROLANDO.

ECKER : sulcus centralis.
BISCHOFF : s. centralis, s. Rolando.
BROCA : scissure de Rolando.
HUXLEY : postero-parietal sulcus.
HENLE : sulcus centralis.
HUGUENIN : fossa Rolandi.
PANSCH (formerly) : fissura transversa anterior.
TURNER, FERRIER, FOWLER : fissure of Rolando.
LUSSANA e LEMOIGNE : scissura di Rolando.
RICHET : scissure de R.
GRATIOLET : sillon de R.

"Die Rolando'sche Furche ist die constanteste und am frühsten auftretende unter den Primärfurchen der lateralen Fläche, und bei den Primaten überhaupt die am meisten charakteristiche." (*Furchen und Wülste, von Pansch,* 1879.) This sulcus makes its appearance in the fifth month of fœtal life. Pansch writes that it is by "Henle: unbennant," but I find it as "sulcus centralis" on page 161 of Henle's "*Nervenlehre.*"

7. SULCUS PARIETALIS.

ECKER: sulcus inter-parietalis.
PANSCH: sulcus parietalis.
BROCA: sillon parietal.
TURNER, HUXLEY, FERRIER: intra-parietal fissure.
RICHET: sillon interparietal.
LUSSANA e LEMOIGNE: scissura intermedia.
VARIOUS: s. postcentralis, s. postrolandicus.

8. SULCUS PRÆCENTRALIS.

PANSCH: sulcus frontalis (posterior division).
ECKER: sulcus præcentralis.
BROCA: sillon prerolandique.
TURNER: ascending limb of sylvian fissure.
FOWLER: vertical frontal fissure.
HUXLEY: antero-parietal.
LUSSANA e LEMOIGNE: solco ascendenti.

I think, in this instance, Ecker's name should be retained, because it expresses its position accurately. Pansch has joined this sulcus to the inferior frontal, just as Turner blended the ascending sylvian ramus with the same sulcus, and, as the older anatomists once caused trouble to their readers by calling the calcarine and occipital fissures "the perpendicular and its branches," the older name, præcentral, should remain. This greatly modifies the divisions into lobules Pansch suggests for this vicinity, and gives us the older designations for them also.

9. SULCUS FRONTALIS SUPERIOR.

PANSCH, ECKER: sulcus frontalis superior.
BROCA, RICHET: premier sillon frontal.
TURNER, HUXLEY, et al.: supero frontal sulcus.
FOWLER: supero-frontal fissure.
LUSSANA e LEMOIGNE: scissura di verga.
VARIOUS: sulcus præcentralis superior, s. prerolandicus.

10. SULCUS FRONTALIS INFERIOR.

PANSCH: sulcus frontalis (anterior division).
ECKER: s. frontalis inferior.

BROCA: sillon second frontal.
TURNER, HUXLEY: infero-frontal sulcus.
FERRIER: inferior frontal.

11. SULCUS TEMPORALIS SUPERIOR.

ECKER: sulcus temporalis superior.
PANSCH: sulcus temporalis.
GALTON'S ECKER: first tempero-sphenoidal.
BROCA: premier sillon temporale.
TURNER: parallel fissure.
RICHET: sillon parallele.
GRATIOLET: scissure parallele.
HUXLEY: superior temporo-sphenoidal f.
FOWLER'S CHARCOT: superior temporal fissure.
LUSSANA e LEMOIGNE: scissura parallela.
BISCHOFF: fissura parallela s. temporalis sup.

12. SULCUS TEMPORALIS MEDIUS.

ECKER: sulcus temporalis medius.
BISCHOFF: fissura temporalis medius.
BROCA: deuxième sillon temporal.
TURNER: second tempero-sphenoidal fissure.
FOWLER'S CHARCOT: middle temporal fissure.
RICHET: represented in wood-cut, but unnamed.

Pansch ignores this sulcus and the succeeding one, but it is too well marked by Leuret and Gratiolet, especially in planche XXI., and in the engraving of the Encephale de Fieschi, planche XXII. It corresponds to the inferior temporal sulcus of the simians, tolerably well marked in the Cynocephalus sphinx, and fully in the macacque.

The omission of either of the lower temporal sulci would leave the included lobules or gyri without boundaries; and as all authors, including Pansch, give three temporal convolutions, this sulcus, in the present condition of our literature, should be retained. It however might be classed in Pansch's Zweifelhafte Primärfurchen.

13. SULCUS TEMPORALIS INFERIOR.

Writers associate this sulcus or sulculus with the preceding, consistently with whichever order they may have adopted, as troisième, inferior, etc. It lies between the preceding and the sulcus collateralis. It is neither so well defined nor so long as the medius, but it is either directly or inferentially mentioned by all but Pansch, who, nevertheless, makes mention of lobules which require it as a boundary. This sulcus might be considered as of doubtful constancy. In any event, it could be compounded of sulculi, and retained until its existence is discredited.

14. SULCUS COLLATERALIS.

PANSCH, ECKER: sulcus occipito-temporalis inferior.
BISCHOFF: fissura collateralis s. temporalis inferior.
HUXLEY: fissura collateralis.
BROCA: quatrième sillon temporal.
TURNER: collateral fissure.
FOWLER: s. occipito-temporalis lateralis.
LUSSANA e LEMOIGNE: scissura occipito-temporale.

This sulcus corresponds in position with the pes accessorius or eminentia collateralis, between the hippocampi, where the posterior and descending cornu join. This relationship being noted by the older anatomists, though they did not name the sulcus, justifies the choice of Huxley, Turner and Bischoff, and certainly has the merit of brevity, which other suggested titles have not.

15. SULCUS OLFACTORIUS.

This is uniformly called olfactory sulcus by all except Broca, who prefers "premier sillon orbitaire." It divides the gyrus rectus from the middle orbital gyrus.

16. SULCUS CALLOSO-MARGINALIS.

ECKER: s. c.-m.
PANSCH: s. c.-m. and s. medialis fronto-parietalis.
BROCA: scissure sous frontale.
TURNER: calloso-marginal fissure.
GRATIOLET: grand sillon du lobe fronto-parietale.
FERRIER, HUXLEY, RICHET: s. c.-m.
LUSSANA e LEMOIGNE: scissura crociata o. parietale interna.

The position of this sulcus above the corpus callosum, beginning under the genu, and coursing below the upper hemispheric margin, ending behind the sulcus Rolando, justifies the name, and is in most favor with anatomists.

17. SULCUS OCCIPITALIS TRANSVERSUS.

FERRIER: s. o. t.

Generally portrayed as a ramus at the lower end of the fissura parietalis. It is important as a means of bounding Gratiolet's *plis de passage* on the occipital extremity.

18. SULCUS ORBITALIS.

ECKER: sulcus orbitalis.
FERRIER: orbital sulcus.
FOWLER: orbital sulcus.
HENLE: indicated.
GRATIOLET: sillon de la face orbitaire du lobe frontal.
LUSSANA e LEMOIGNE: solco orbitale.

While Pansch omits this, the Italians designate one in addition. Most authors divide the orbital lobe into three convolutions, the boundaries of which are determined by this and the olfactory sulcus.

19. Sulcus occipitalis longitudinalis inferior.

This small sulcus is between the second and third occipital gyri. *Occipitalis minor* might be more suitable; but abstaining from any contribution to the overburdened vocabulary, I give Ecker's choice.

Sulculus paracentralis transversus.

A constant furrow found in the paracentral gyrus of Betz, and called by him the transverse furrow of the paracentral lobule.

PRIMARY OR CONSTANT CONVOLUTIONS.

A. EXTERNAL SURFACE.

I. Lobulus frontalis superior.

Pansch: gyrus frontalis superior.
Ecker: erste Stirnwindungsgruppe, gyrus frontalis superior.
Henle: oberster gyrus frontalis.
Huxley: gyrus supero-frontal.
Broca: première circonvolution frontale.
Turner: superior frontal gyrus.
Huguenin: dritte stirnwindung.
Valentin: gyrus adplicatus anterior.
Lussana e Lemoigne: circonvoluzione parietale anteriori.
Ferrier: superior frontal convolution.
Fowler's Charcot: first frontal convolution.
Richet: première circonvolution frontale.
Gratiolet: première circonvolution frontale.

II. Lobulus frontalis medius.

Pansch: gyrus frontalis medius.
Bischoff: zweite Stirnwindungsgruppe.
Henle: mittlerer gyrus frontalis.
Broca: deuxième circonvolution frontale.
Turner: middle frontal gyrus,
Huguenin: zweite Stirnwindung.
Lussana e Lemoigne: circonvoluzione circolare.
Ferrier: middle frontal convolution.
Fowler: second frontal convolution.
Gratiolet, Richet: deuxième circonvolution frontale.
Huxley: gyrus medio-frontal.

III. Lobulus frontalis inferior.

Ecker: gyrus frontalis tertius s. inferior.
Bischoff: dritte Stirnwindungsgruppe.
Henle: unterer gyrus frontalis g. transitions (nach Huschke).
Broca: troisième circonvolution frontale.
Turner: inferior frontal gyrus.

Huguenin: erste Stirnwindung.
Lussana e Lemoigne: circonvoluzione spirale.
Ferrier: inferior frontal convolution. Broca's conv.
Fowler: second frontal convolution. Broca's conv.
Gratiolet, Richet: troisème circonvolution frontale.
Huxley: gyrus infero-frontal.

IV. Lobulus Rolandicus anterior.

Pansch: gyrus Rolandicus anterior.
Ecker: gyrus centralis anterior.
Bischoff: vordere Centralwindung.
Henle: gyrus centralis anterior.
Broca: circonvolution prerolandique.
Turner: ascending frontal gyrus.
Fowler: ascending frontal convolution.
Ferrier: anterior central convolution.
Huguenin: vordere Centralwindung.
Lussana e Lemoigne: circon. parietale anteriori.
Valentin: gyrus adplicatus anterior.
Richet: circon. frontale ascendente.
Pozzi: quatrième circon. frontale.
Rolando: processi anteroidei verticali di mezzo (partie anteriore).
Huxley: gyrus antero-parietal.
Gratiolet: première pli pariétal ascendant.

The division of the preceding group is conformable to the majority of authors. Pansch, in consequence of joining the præcentral and second frontal sulci, re-arranged these convolutions on rather an inconvenient plan. His names, however, for the portions contiguous to sulcus Rolando, are very expressive.

V. Lobulus Rolandicus posterior.

Pansch: gyrus Rolandicus posterior.
Ecker: g. centralis posterior.
Bischoff: hintere Centralwindung.
Henle: g. centralis posterior.
Broca: circonvolution postrolandique.
Turner: ascending parietal gyrus.
Huguenin: hintere Centralwindung.
Lussana e Lemoigne: circon. parietale posteriore lunga.
Valentin: gyrus adplicatus posterior.
Richet: parietale ascendente circon.
Ferrier: posterior central convolution.
Fowler's Charcot: ascending parietal convolution.

VI. Lobulus parietalis superior.

Pansch: gyrus parietalis superior.
Ecker: lobulus parietalis superior.
Bischoff: Vorzwickel und innere obere Scheitelbogenwindung.

HENLE: præcuneus.
BROCA: première circon. pariétale.
TURNER: postero-parietal lobule.
FERRIER: superior or postero-parietal lobule.
FOWLER: superior parietal lobule.
RICHET: lobule pariétal superieur.
HUGUENIN: dritte Scheitelwindung.

VII. LOBULUS PARIETALIS INFERIOR.

PANSCH: lobulus parietalis inferior.
ECKER: lobulus p. inf., a. lob. supramarginalis, b. gyrus angularis.
BISCHOFF: vordere und mittlere Scheitelbogenwindung.
HENLE: lobulus tuberis.
BROCA: deuxième circonvolution pariétale.
TURNER: lobule of the supra-marginal convolution and angular gyrus.
RICHET: lobule parietal inferior et pli courbe.
FERRIER: inferior parietal lobule, gyri supramarg. et ang.
LUSSANA e LEMOIGNE: lobo quadrangolore.
HUGUENIN: erste und zweite Scheitelwindung.
FOWLER: inferior temporal lobule, lobulus parietalis inferior et gyrus angularis.

One of the advantages of distinguishing between gyrus and lobulus is apparent in this convolution, where some authors locate two lobules in one gyrus, two gyri in one convolution or lobule, and even a lobule and gyrus in a convolution called a gyrus.

VIII. LOBULUS TEMPORALIS SUPERIOR.

PANSCH: lobulus temporalis superior.
ECKER: gyrus temporalis superior.
BISCHOFF: erste obere Schläfenwindung.
HUGUENIN: erste Schläfenwindung, gyrus temporalis I.
BROCA, RICHET: première circon. temporale.
TURNER: superior temporo-sphenoidal convolution.
FERRIER: first temporo-sphenoidal convolution.
LUSSANA e LEMOIGNE: prima circonvoluzione temporali.
FOWLER: first temporal convolution.

IX. LOBULUS TEMPORALIS MEDIUS.

PANSCH: upper half of lobulus temporalis inferior.
ECKER: gyrus temporalis medius.
BISCHOFF: zweite mittlere Schläfenwindung.
HENLE: unnamed, but indicated.
BROCA: deuxième circonvolution temporale.
TURNER: middle temporo-sphenoidal convolution.
HUGUENIN: zweite Schläfenwindung, gyrus temporalis II.
LUSSANA e LEMOIGNE: secondo circon. temporali.
GRATIOLET: pli temporal moyen.
VALENTIN: supplementum gyri anguiformis post. et inf.

As previously mentioned under Sulci, Pansch unites this lobule with the following under the name of lobulus temporalis inferior, which is not done by any other investigator.

X. LOBULUS TEMPORALIS INFERIOR.

PANSCH: gyrus temporalis lateralis.
LUSSANA e LEMOIGNE: terza circon. sfeno-temporali.
GRATIOLET: pli temporal inferiéur.
ROLANDO: processo semi duplicato del lobo di mezzo.

The others invariably follow the order indicated above, with the substitution of troisième, inferior or third as prefix. Pansch attaches this as a gyrus to the lobulus occipito-temporalis lateralis.

B. MEDIAN SURFACE.

XI. LOBULUS MARGINALIS.

PANSCH: gyrus medialis fronto-parietalis of the lobulus medialis anterior.
ECKER: g. frontalis superior, mediale Fläche.
BISCHOFF: Innenfläche des ersten Stirnwindungszuges und obere Bogenverbindung der Centralwindungen.
BROCA: première circon. frontale.
TURNER: marginal gyrus.
RICHET: face moyenne de la première circon. frontale.
LUSSANA e LEMOIGNE: cintura periferica.
HUXLEY: marginal convolution.

The title Turner gives this portion seems as expressive as any, and decidedly shorter.

XII. LOBULUS FORNICATUS.

PANSCH; gyrus cinguli of the lobulus medialis anterior.
ECKER: gyrus fornicatus und præcuneus.
BISCHOFF: gyrus fornicatus.
BROCA: grande circonvolution du corps calleux.
TURNER: conv. of corpus callosum and quadrilateral lobule.
HUGUENIN: gyrus fornicatus.
FOWLER: conv. of corpus callosum.
LUSSANA e LEMOIGNE: processo quadrilatero e circonvoluzione madre-interna-dell' orletto.
VALENTIN: circonvolutio cristata.
RICHET: præcuneus et gyrus fornicatus.

The ancient title, fornicatus, refers to the arched shape of this lobule, and not to its being near the fornix, and by this name the convolution is generally known to anatomists. For this reason it should be retained.

XIII. LOBULUS CUNEUS.

PANSCH: lobulus medialis posterior, gyrus medialis occipitalis s. cuneus.
ECKER: gyrus cunei.

Henle: cuneus.
Broca: lobule triangulaire.
Turner: cuneus or occipital lobule.
Bischoff: Zwickel.
Lussana e Lemoigne: circon. angolare occipitale.

XIV. Lobulus occipito-temporalis medialis.

Pansch: lobulus occipito-temporalis medialis.
Ecker: gyrus occipito-temporalis medialis, gyrus Hippocampi.
Bischoff: unterer innerer Hinterhauptwindungszug und gyrus Hippocampi.
Huschke: lobulus lingualis.
Henle: gyrus Hippocampi.
Huguenin: Zungenwindung.
Broca: cinquième circonvolution temporale.
Turner: uncinate gyrus, middle internal temporal gyrus.
Richet: gyrus hippocampi et g. uncinatus et lobulus lingualis.
Ferrier: g. uncinatus, uncus gyri fornicati, subiculum cornu ammonis and crochet with gyrus occipito-temporalis medialis (lobulus lingualis).
Fowler's Charcot: Unciform and hippocampal convolutions.

By including all that portion of the brain lying beneath the fissura calcarina from the occipital to the temporal extremities into one lobule, bounded below by the fissura collateralis, and designating the smaller important points as gyri, avoids much mislocation and confusion. While there has been perfect agreement as to portions, hereabouts, there has been total want of unanimity as to terms, the same names being applied to different parts. The designation of Pansch is complete. On page 58 of Fowler's Charcot, in Fig. 17, representing the vascular territories of the inferior face of the human brain, the median and lateral temporo-occipital convolutions are erroneously transposed.

C. BASILAR SURFACE.

XV. Lobulus occipito-temporalis lateralis.

Pansch: gyrus occipito-temporalis lateralis.
Ecker: gyrus occipito-temporalis lateralis.
Bischoff: unterer äusserer Hinterhauptwindungszug und ein Theil der dritten Schläfenwindung.
Huschke: gyrus fusiformis.
Broca: quatrième circonvolution temporale.
Turner: inferior internal temporal gyrus.
Ferrier: gyrus occipito-temporalis lateralis (fusiformis).
Richet: g. occipito-temporalis, lobulus fusiformis.
Lussana e Lemoigne: processo fondamentale posterior.
Fowler's Charcot: oc-temp. lat. convolution (medialis, p. 58, error).
Huguenin: Spindelwindung.

XVI. LOBULUS ORBITALIS.

Pansch does not name the sulcus between the middle and lateral gyri in this lobule. All others do, and one adds a sulcus too many. The name of this lobule is uniformly the same among authors.

GYRI.

EXTERNAL SURFACE.

a. GYRUS OPERCULUM.

GRATIOLET: pli surcilier.

VARIOUS: Operculum; klappdeckel; opercule; l'opercolo; coperchio; convolution of Broca; circonvolution spirale; roof of Island of Reil.

b. GYRUS SUPRA-MARGINALIS.

GRATIOLET: lobule du pli courbe.

VARIOUS: supra-marginal lobule or gyrus.

LUSSANA e LEMOIGNE: circonvoluzione marginale.

HUSCHKE: lobulus tuberis.

c. GYRUS ANGULARIS.

GRATIOLET: pli courbe.

FERRIER, HUXLEY: angular gyrus.

Gyri *b* and *c* are not separate in the ape.

d. GYRUS OCCIPITALIS PRIMUS.

e. GYRUS OCCIPITALIS SECUNDUS.

f. GYRUS OCCIPITALIS TERTIUS.

These are the three "bridging convolutions" or "plis de passage" to which term Ecker objects, calling them first, second and third occipital gyri, as above. Huxley writes of them as annectant external gyri. Lussana and Lemoigne call them "gyri abrupti e cuneiformes, piega occipitale de passagio."

MEDIAN SURFACE.

g. GYRUS UNCINATUS.

Usually mentioned with many names by each writer: gyrus uncinatus; crotchet; gyrus Hippocampi; uncus gyri fornicati; subiculum cornu ammonis; cornu ammonis; the hook or reduplication of the uncinate gyrus.

h. GYRUS DENTATUS.

The hippocampal gyrus of some authors. The fascia dentata bordering the hippocampus major, or investing *corps godronné* suggesting the title.

j. GYRUS PARACENTRALIS.

Named by Betz, who, with Mierzejewski, discovered here the presence of comparatively enormous pyramidal cells, called "giant cells" from their size. Ferrier locates in this vicinity, the psycho-motor centres of the limbs. A sulculus is noticed in this gyrus, mentioned as the transverse furrow of the paracentral lobule.

k. GYRUS PRÆCUNEUS.

The quadrilateral lobule or præcuneus: avant coin; Vorzwickel. Considering the Greek origin of the word gyrus, quadrilateral gyrus, or four-sided circle would amount to an etymological squaring of the circle.

l. GYRUS DESCENDENS.

Between the rami of the fissura calcarina.

BASILAR SURFACE.

m. GYRUS RECTUS.

n. GYRUS ORBITALIS MEDIUS.

o. GYRUS ORBITALIS LATERALIS.

INSULA.

Island of Reil, which might be classed among the lobules as lobulus insulæ. Ecker prefers "lobus centralis." Spitzka suggests a suitable nomenclature applicable to comparative encephalic anatomy, in the JOURNAL OF NERVOUS AND MENTAL DISEASE, July, current series. Spitzka's article is a valuable addition to our literature and knowledge of the insula. He objects to naming the shallow groove of a turtle's or serpent's brain, island or fossa, although homologous with the insula, and uses the term "fovea centralis," which applies to all true reptiles and birds; "fossa centralis" for its further development in rodentia, insectivora and marsupialia; "lobus centralis" where the surrounding convolutions overlap the retracted area. The Insula contains the three GYRI BREVES or OPERTI.

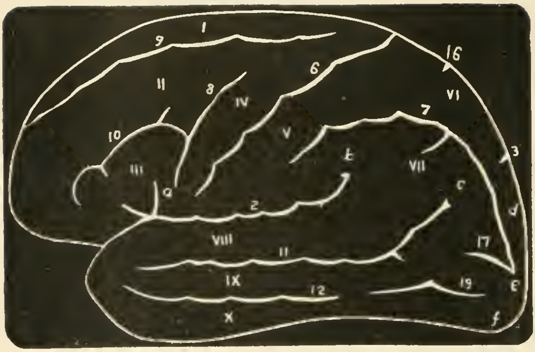
They lie within the lips or borders of the fissura Sylvii concealed by the gyrus operculum, which on its under surface contains gyri dovetailing between the gyri breves *in situ.*

The insula may also be described as lying between the operculum externally and the extra-ventricular nucleus of the corpus striatum internally.

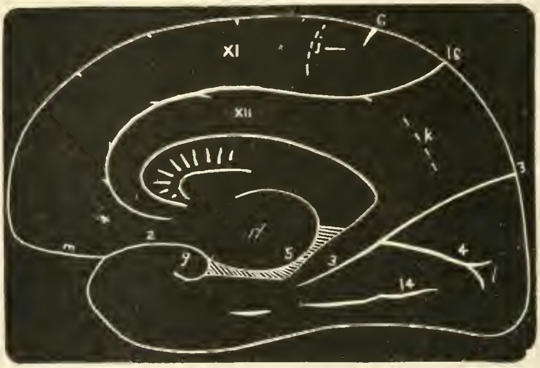
Gratiolet et Leuret, planche XVII., illustrate this "central lobule" or "plis rayonnants de l'Insula" as having four gyri or "*circonvolutions disposées en manière d' éventail et se continuant avec les plis superficiels de l' hémisphère.*

I would suggest from their positions for the principal or more evident three: *Gyri breves: anterior, medius, posterior.*

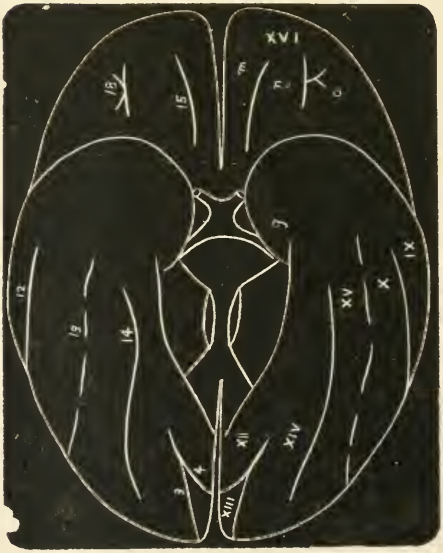
Where extra gyri require mention we happily have a means of clearly designating them. The typical middle cerebral or Sylvian artery at this point divides into four branches, which lie in the sulci separating the gyri breves. It is evident that



Cut A. External Surface.



Cut B. Median Surface.



Cut C. Basilar Surface.

four arterial branches can include but three gyri, hence additional undulations must be attached, or referable in some way, to these three principal divisions.

POSITIONS.

In the three preceding cuts the Roman numerals refer to the lobules as numbered, *seriatim*, in the paper. The Arabic numerals are intended to represent the fissures, up to five, and thereafter the sulci; the "lower case" letters are placed over the gyri; and each number and letter may be referred to under respective headings: Lobules, Fissures, Sulci, Gyri.

It would be out of place and presumptuous to attempt a digest of what has been done in the field of localization in a simply topographical essay, particularly as the topography is what the term implies — a simple survey of the outer configuration of the cortex of the cerebrum. Still, I think it would be practicable and interesting to note, *en passant*, such matters of physiological, anatomical or pathological importance as have been evolved by the exact, cautious and conscientious experimenters and observers of many nationalities. It is necessary, then, to resume and locate as clearly as possible each furrow and convolution:

1. *Fissura longitudinalis.*—Divides the cerebrum into two hemispheres, antero-posteriorly, except where the right and left lobes are joined by the corpus callosum, the great transverse commissure which links the two sides symmetrically into a physiological whole. To exhibit the entire median surface, an incision is carried downward between the anterior cerebral arteries, through the corpus callosum; septum lucidum, exposing the fifth ventricle; fornix, when the Venæ Galeni and velum interpositum come into view, with the anterior commissure behind a band of fibres, connecting the genu of the corpus with the optic chiasma, the dipping anterior pillar of the fornix blending downward to its corpus mammilarius. Behind this the foramen of Monro and upper part of the thalamus opticus, with the pineal gland, appear. Carrying the incision down about two lines, the middle or soft commissure is cut through, and the connected posterior commissure, pineal gland, and quadrigeminal body are divided,

the scalpel falling into the aquæductus Sylvii and third ventricle.

Dividing the optic decussation and pituitary body, completes the separation, and we have the median surface exposed.

16. *Sulcus calloso-marginalis.*—Begins under the genu of the corpus, and running nearly parallel with the upper margin of this great band of fibres, courses posteriorly, and ends as a notch on the lateral parietal surface.

5. *Fissura hippocampi.*—Runs between the posterior border of the fornix to the hook or crotchet, which forms the gyrus uncinatus in the temporal lobe.

3. *Fissura occipitalis.*—Begins behind the last mentioned, with which it is confluent in the ape, but separately existent in man, runs directly and nearly perpendicularly backward, bending over the latero-median border, and ending as does the calloso-marginal sulcus. In the ape it is prolonged on the occipito-parietal lateral surface.

4. *Fissura calcarina.*—Branches from the last, near its anterior extremity, and passing horizontally backward, parts into two rami, ascendens et descendens, on the occipital extremity.

14. *Sulcus collateralis.*—Lies under, and somewhat parallel to, the preceding, marking externally the position of the eminentia collateralis, and separating the posterior portion of the median from the basilar surface.

XI. *Lobulus marginalis.*—Is bounded by the latero-median crest, anteriorly and superiorly, and by the sulcus 16, posteriorly and inferiorly. Just anterior to the median termination of the sulcus Rolando in this lobule are the *sulculus paracentralis transversus* and the *gyrus paracentralis*, which surrounds it.

XII. *Lobulus fornicatus.*—Directly under XI. and above the corpus callosum, which, with 3 and 5, form its lower boundaries, the latero-median ridge completing the posterior separation from other lobules.

The posterior portion of XII. includes the *gyrus præcuneus*, a quadrangular part separated by sulculi from the lobule proper.

XIII. *Lobulus cuneus.*—A triangular portion of the occipital extremity, included between the fissures 3 and 4. Regarding the ramus descendens as the nethermost part of 4, causes the *gyrus descendens* to be located in XIII.; though in many brains it may apparently be a portion of the lobule immediately below.

XIV. *Lobulus occipito-temporalis medialis.*—Extends from the occipital to the temporal extremity, beneath the fissures 2, 5 and 4. Accessory sulculi have to be called into requisition to complete the lower boundary, as the sulcus collateralis does not extend into the temporal extremity. An important *gyrus*—the *uncinatus*—formed like a hook, is found near the anterior perforated space in this lobule.

2. *Fissura Sylvii.*—Begins at the gyrus uncinatus, and separating the hemisphere into upper and lower portions or lobes, the frontal and temporal runs nearly horizontally backward to the postero-parietal part of the lateral surface, where it ends in the *gyrus supra-marginalis;* within this fissure, and just beneath the *gyrus operculum*, formed by the *ascending ramus* of the fissura Sylvii and sulci posterior to it, lie the *gyri breves, anterior, medius et posterior*, previously described as belonging to the *insula* or *island of Reil.*

6. *Sulcus Rolando.*—Begins at the median edge, superiorly, about midway between parts corresponding to the anterior and posterior borders of the parietal bone, thence vertically forward and downward, nearly to the fissura Sylvii.

9. *Sulcus frontalis superior.*—Commences anterior to 6, passes forward nearly parallel to 1, tending toward the median surface, probably, in most brains, and breaks up into sulculi on the superior orbital extremity.

10. *Sulcus frontalis inferior.*—Either as a continuation of 8, or at right angles to it, nearly parallel to the previously mentioned sulcus 9, ends similarly in sulculi, usually on the latero-posterior part of the orbital extremity.

8. *Sulcus præcentralis.*—A short sulcus, anterior and running nearly parallel to 6, ending in, or passing at, right angles to 10.

7. *Sulcus parietalis.* — On the superior postero-parietal region, for a short space in the same direction with 6, but bends downward and to the median line, ends on the occipital

extremity, a little below where fissure 3 emerges from the median surface.

11. *Sulcus temporalis superior.*—From the temporal extremity backward, parallel to fissure 2, ends in the *angular gyrus*, just short of sulcus 7, at the occipito-parietal junction. This sulcus and fissure 2 are confluent and identical, posteriorly, in the simian; hence, the two gyri formed by them in the human brain, form but one in the ape's.

12. *Sulcus temporalis medius.*—Below and parallel to the preceding anteriorly, but ends abruptly in sulculi, before reaching the occipital extremity.

17. *Sulcus occipitalis transversus.*—Might be considered, and probably is, in many instances, a ramus projected forward from the occipital termination of sulcus 7.

19. *Sulcus occipitalis longitudinalis inferior.*—A shorter sulcus than its name would seem to indicate, with as much disparity between size and title as exists in musculus levator labii superioris alæque nasi. This sulcus is on the infero-lateral occipital extremity below sulcus 17, and could be made continuous with 12 by means of intervening sulculi.

I. *Lobulus frontalis superior.*—Anterior to sulcus 6, and superior to 9, extends to orbital extremity.

II. *Lobulus frontalis medius.*—Bounded anteriorly by orbital extremity, posteriorly by sulcus 8, above and below by sulci 9 and 10.

III. *Lobulus frontalis inferior.*—Between sulcus 8 posteriorly, 10 superiorly, fissure 2, and orbital extremity infero-anteriorly.

IV. *Lobulus Rolandicus anterior.*—In front of the sulcus 6, behind sulcus 8, and separated from lobules I. and II. by sulculi or imaginary lines.

V. *Lobulus Rolandicus posterior.*—Behind sulcus 6, separated from posterior lobules by fissure 2 and sulculi projected superiorly from it into the sulcus 7, and thence toward the median edge.

VI. *Lobulus parietalis superior.*—Posterior to V., and between sulcus 7 and latero-median edge or margin, generally described as ending at the point of emergence of fissure 3, but, to avoid multiplying lobules on the occipital extremity, continned to the end of sulcus 7, leaving the *gyrus occipitalis primus* included in this lobule at its posterior border.

VII. *Lobulus parietalis inferior.*—Beneath the sulcus 7 from V. to sulcus 17. I would recommend omitting this lobule altogether in future, and leaving off the word superior from its companion lobule above. Lobules VIII. and IX. could thus include all the parietal portion up to sulcus 7. As it is, an imaginary line running at right angles to the ends of sulcus 11 and fissure 2 must be projected to meet sulcus 17, which, by the plan mentioned, would be avoided. Still greater ambiguity in the location of this lobule exists in authors projecting it anteriorly into lobule V., from which it can be divided by nothing but sulculi.

VIII. *Lobulus temporalis superior.*—Between fissure 2 and lobulus VII., superiorly and sulcus 11 inferiorly, ends anteriorly in temporal extremity.

IX. *Lobulus temporalis medius.*—Bounded above by sulcus 11, postero-inferiorly by lobulus VII. and sulcus 12, and sulculi extending to sulcus 19.

X. *Lobulus temporalis inferior.*—Extends from the temporal to the occipital extremity, lying between the sulci 12 and 13 in front, and 19 and sulculi projected backward from 13 behind.

The gyri remaining to record on the lateral surface are the three on the occipital extremity:

Gyrus occipitalis primus at the lower end of the lobule VI.

Gyrus occipitalis secundus.—Between sulci 17 and 19.

Gyrus occipitalis tertius.—Below sulcus 19, occupying the most extreme part of the occipital region.

Posteriorly, these gyri are bounded by fissura 1 or the latero-median margin.

Upon the base of the brain, the remaining points are:

13. *Sulcus temporalis inferior.*—Midway, parallel to and co-extensive with sulci 12 and 14.

15. *Sulcus olfactorius.*—Immediately above the olfactory nerve and bulb.

18. *Sulcus orbitalis.*—Antero-posteriorly on the orbital surface of the orbital extremity, usually with one or more rami.

XV. *Lobulus occipito-temporalis lateralis.*—Between sulci 13 and 14 and extremitates temporalis et occipitalis.

XVI. *Lobulus orbitalis.*—Anterior to temporal extremity on the infero-orbital surface.

m. Gyrus rectus.—Between median margin and sulcus olfactorius.

n. Gyrus orbitalis medius.—Between orbital and olfactory sulci.

o. Gyrus orbitalis lateralis.—Above and external to sulcus orbitalis, blends with lobulus III. on lateral surface and orbital extremity.

The preceding details of cerebral topography warrant the very great labor and the vast amount of polemics, of the numerous investigators in this department of anatomy, in their application to autopsies alone. The inclination, skill and opportunities to make accurate *post-mortem* records of cerebral lesions has existed among surgeons ever since surgery was worthy of the name, but this topographical lore has been slowly evolved, and is slow to find its way into anatomical treatises. Without deploring the opportunities lost by Erasistratus, while dissecting his criminals alive, to have placed the science of localization (Fritsch, Hitzig and Ferrier have made it a science) ages ahead of its present slow but sure progress, we could conceive the value of accurately investigated and recorded cerebral injuries, with *ante-mortem* phenomena, statistically set forth in such works as the Surgical Reports of the late war. But the value of the study becomes more apparent in the revelations made by electrization of the cortex, as practiced upon the lower animals by Fritsch and Hitzig, who, in 1870, published in Reichert and DuBois Reymond's *Archiv*, the results of their experiments, which Ferrier, a little later, verified and announced in English journals. The latter investigator improved upon the method of galvanic stimulation used by the originators, in using the secondary or induced current of DuBois Reymond's inductorium, of the mean battery strength of one Daniell. The primary coil resistance was 1.9 units. Ferrier does not say which unit he means, Siemen's or Ohm's. The former is used largely on the European continent, the latter was established by the British Association. To convert Siemen's into Ohm's, multiply by .9536; Ohm's into Siemen's, 1.0486. This may seem a little hypercritical, but it would have been as well had Ferrier noted which was used. However, at a distance of 8 cm. between the two coils, he obtained a current with a "pungent but bearable taste," when applied to the tip of the tongue. It was this current from blunted electrodes applied to the cortex with which the motions, etc., of the animals were evoked. Misunderstandings and assertions that such currents would be diffused, and criticisms upon his methods finally led Ferrier to inveigh against fixing any standard of galvanization beyond using enough of the secondary *to produce the effects uniformly attainable.* The matter of extra polar conduction was settled in Ferrier's favor.

By the inductive method of reasoning, very much praised and too seldom used, Hughlings Jackson was inspired, before either of these three workers, with the idea he expressed in *Clinical and Physiological Researches on the Nervous System*, that convulsive movements of one side might be due to localized irritation in the opposite hemisphere, and inferred a "discharging" lesion from the cortex. His confreres considered him fanciful and ridiculed his theory. The main points discovered are as follows:

The antero-frontal and occipital regions gave no response to electrical stimulation. The application of the electrodes to the insula, lobulus fornicatus and corpus callosum were similarly negative in effect. The lobulus marginalis coincided in phenomena with the frontal lobule bordering it.

By homology of centres surrounding it, localized by this means, the crucial sulcus of the carnivores corresponds with sulcus Rolando *physiologically.* Anatomically the two are not capable of comparison. It is noteworthy that the sigmoid gyrus at the end of this crucial sulcus is situated in all carnivora far in front in the orbital region superiorly. The significance of this fact seems to have escaped the notice of authors. It is a conveyance forward of psycho-motor centres into a region corresponding to inhibitory functions in the human brain; inhibition appearing to Ferrier to be "a fundamental element in the attentive concentration of consciousness and control of ideation."

As it is neither desirable nor convenient to copy Charcot and Ferrier into these pages, completely and verbatim, I shall try to epitomize interesting appropriate points only.

Behind the parietal ends of sulci 6 and 16 in lobule VI. are situated centres for movements of the opposite leg and foot, such as are concerned in locomotion.

In front of this, extending across sulcus 6 about 2 cm. in the posterior part of lobulus I., various complex movements of the arms and legs as in swimming, climbing, etc. In the monkey the lower part of this area is devoted to tail movements. The evolutionists might look here for apolar or rudimentary cells.

About 2 cm. further forward extends an area devoted to arm and hand extensors, as in reaching forward.

Behind the orbital extremity on lobule I., 3 cm. in diameter, lying partly on lobule II., is a centre for lateral movements of head and eyes, and dilatation of pupil.

Below the posterior end of sulcus 9 in lobulus IV., bicipital supination and flexion of forearm. Thence three centimetres down the same lobulus, centres for elevation and depression of angle of mouth.

On the gyrus operculum, movements of lips and tongue as in articulation. This is the region disease of which causes aphasia, and known as Broca's convolution.

At the lower end of lobulus V., the centre of the platysma, retraction of angle of mouth. Thence up to the first point mentioned on lobulus V. to lobulus VI., centres for hand and wrist movements.

The supra-marginal and angular gyri indicate centres of vision.

Beneath these and upon the posterior portion of lobulus VIII., centres of hearing.

The centre of smell is in the gyrus uncinatus; near it but not exactly defined is a taste centre.

Touch is situated in the middle of lobulus XIV., near fissure 5.

A region "in close relation to the centres of smell and tactile sensation might be regarded as the probable seat of sensations forming the basis of sexual appetite." The occipito-temporal lobules on the basilar surface might fulfill the conditions.

Visceral sensations in the occipital extremity; ablation causing anorexia.

Merely mentioning that corroborative experiments were instituted, by vivisection, to supplement the information obtained through electrical stimulation, we will pass on to the vascular supply.

Heubner and Duret, among the moderns, have done more to forward knowledge of the peculiarities of blood-supply to the brain than any; while Charcot has worked in the field of lesions arising from hemorrhages, embolisms, and softenings producing pareses, paralyses, etc.

We are all familiar with the circle of Willis at the base of the brain, but the books do not follow beyond the main or primary branches given off from the carotid and basilar arteries.

The *anterior cerebral* artery passes into the median surface, runs backward over the corpus callosum, and anastomoses with the posterior cerebral in the occipital region. It gives off three principal branches: the first is distributed to the orbital extremity, the second to the lobulus fornicatus, corpus callosum, marginal lobule, first and second frontal lobules, and gyrus paracentralis. The third branch goes to the gyrus præcuneus, which may be subject to lesions thus on its own account. The *middle cerebral*, or *Sylvian* artery divides into four principal branches, a lesser branch at its origin supplying the central ganglia. The main trunk runs to the Insula, and at that point divides and emerges on the lateral or external face of the brain, between the Sylvian borders. The first branch Duret calls the frontal external and inferior. As it supplies the operculum, many instances of aphasia from its obliteration are on record.

The second branch is the anterior parietal, going to lobulus Rolandicus anterior.

The third ascends the posterior Rolandic lobule.

The fourth branch goes to the gyrus angularis and lobulus temporalis superior.

Softenings in the regions supplied by these arteries have produced the uniform consequences consistent with the function localized in each part. The alteration in the anterior and posterior Rolandic lobules have been invariably followed by paralyses, when the basal ganglia were intact. A softening of the gyrus præcuneus has been followed by nothing noticeable, consistently with Ferrier's experiments.

The middle cerebral is much more frequently the seat of serious alterations than the anterior cerebral artery, "doubtless in part owing to the angle at which the sylvian leaves the internal carotid artery."

"The *posterior cerebral artery* often suffers alterations by embolismus and thrombus.

"Ischæmic softenings of the posterior lobes are much more common than with the anterior lobes."—Charcot, p. 58. Three departments are supplied by this artery: The gyrus angularis; inferior part of the temporal lobe; and lobulus occipito-temporalis lateralis; the third division contains the lobulus occipito-temporalis medialis, lobulus cuneus and occipital extremity, generally.

It is noteworthy that the gyrus angularis receives blood-supply from two sources, which would tend to lessen the chances for amblyopia or hemiopia from cortical lesions.

The literature of this subject cannot be fairly discussed in this article, so with the foregoing mere outlines I will close.

With the desire to "render unto Cæsar," I will repeat that to Pansch I am indebted for the basis of this scheme, and have only departed from his system where other authorities, his collaborators, took issue with him, and their views seemed better.

Wherever an author could be quoted verbatim I have preferred doing so, but generally found it necessary to change the phraseology in condensing.

To Prof. Jewell and Dr. Bannister I am indebted for valuable suggestions and other assistance, and am under especial obligations to the former for the free use of his excellent and extensive library, as well as for having revised this essay. But it is in better taste to thank him for his careful instruction during my pupilage under him, on my own account and for my classmates.

ART. VII.—A NEW FUNCTION OF THE CORPORA QUADRIGEMINA.

BY G. B. WOOD FIELD, STUD. MED., AND ISAAC OTT, M. D.

THAT the secretion of sweat is under the domination of nerve centres seated in the spinal cord and medulla oblongata has been proved by Luchsinger, Nawrocki, Vulpian[*] and others. One of us, with Woroschiloff's instrument, has proven that in the cat the sweat fibres run in the lateral columns of the spinal cord; that the cells of the sweat glands exhibit microscopical changes corresponding to the activity of the secretory process, the cells in activity being smaller than the cells at rest, more granular as regards their protoplasmic contents, and more highly tinged by carmine; that there is a peripheral inhibitory apparatus of the sweat secretion, for when pilocarpin is administered irritation of the sciatic checks the secretion, as well as local irritation applied to the ball of the foot; that probably the multipolar ganglion cells, described by Coyne, on the sweat glands, are a part of the secreto-inhibitory apparatus; that after section of the sciatic the secretory nerves live longer than the motor; that after death of the secretory nerves muscarin is still able to call the sweat glands into activity, which pilocarpin is not able to do. Vulpian[†] has already located an apparatus inhibiting sweat in the medulla oblongata; but irritation of this part by electrical currents always brings out a profuse secretion of sweat, and sensory irritation is not able to arrest it. Our aim has been to discover the seat of the inhibition of sweat, for several pathological cases seemed to point to the existence of such a mechanism.

Method:—Young cats were selected as the most suitable for these experiments, as they sweat very readily. Then, after the method described by Nothnagel, a concentrated solution of chromic acid was injected by means of a hypodermic syringe into various parts of the brain. In young animals the needle penetrates the skull without the need of a trephine. The sweat secretion was noted before the operation and for some hours or days after the injection. When injections were made into the upper part of the medulla oblongata no inhibition of sweat was observed, neither when the lesion involved the pons varolii or the cerebellum. But when the corpora quadrigemina were affected by it then the feet became very dry and remained so for some days. The seat of lesion by the acid was discovered by removing the skull with a bone forceps, hardening the brain in alcohol, and then removing portions of it till the discolored broken down spot in the brain was discovered. The following experiment, being but one of many, demonstrates the secreto-inhibitory centre: Ex. 1.—Cat, etherized; injection of a few drops of a solution of chromic acid into the corpora quadrigemina (as proved on autopsy). After the animal recovered from the shock, the feet were examined and found to be quite dry, being very moist previous to the injection. This dryness of feet continued for some days.

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[*] *Journal of Physiology*, Vol. II., No. 1, 1879.

[†] *Comptes Rendus*, tome LXXXVI., p. 1276.

That the arrest of secretion is not due to vaso-motor changes is proved by the fact of this centre not being located near the point of irritation by the acid. Further, one of us has proven by experiments, that circulatory changes have hardly any effect at all upon the sweat-secretion. That the sweat glands are still able to secrete is proven by the production of asphyxia, the carbonic anhydride here acting as an excitant. This method of experimentation excludes any errors which may be due to spread of current when electricity is used. That electric irritation will also arrest the secretion of sweat was proved by the following experiment:

Ex. 2.—Kitten; received subcutaneously about a cubic centimetre of fluid extract of jaborandi—two holes were trephined into the skull. When the animal was sweating profusely from the drug, needle electrodes insulated to near their points were sunk into the brain so as to irritate the corpora quadrigemina. A very feeble current was used, derived from a DuBois induction apparatus. The sweating was arrested, and this arrest continued for some time after the irritation was removed.

The fact of an inhibitory apparatus located in the tubercular quadrigemina explains how we have a hot yet a non-sweating skin. Heat is normally a great excitant of the sweat centres, but in fever the poison irritates the inhibitory apparatus of the secretion of sweat. Fright not only paralyzes the cardio-inhibitory apparatus, but also the suduro-inhibitory apparatus, whereby the spinal sweat centres come into full play. Feeble persons, whose inhibitory mechanisms are always weak, perspire easily for similar reasons.

Schuh also relates two cases where after section of the frontal nerve, sweating was continuous on that side of the brow ever afterwards.

Further proof with the experiments in detail will be presented by one of us (Mr. Field) in his thesis.

American Neurological Association.

FIFTH ANNUAL MEETING.

WEDNESDAY, JUNE 18TH—FIRST DAY—AFTERNOON SESSION.

The Association was called to order at 3 p. m., by the Secretary, Dr. E. C. Seguin, of New York, who introduced the President, Dr. F. T. Miles, of Baltimore, Md.

Present—Drs. Beard, Edes, Gray, Hammond, McBride, Mason, Miles, Putnam, Rockwell, Schmidt, Seguin, Shaw and Spitzka.

The President thanked the Association for the honor it had conferred upon him in his absence, and proceeded with the regular order of business.

On recommendation of the Council, the resignations of the following gentlemen were accepted: Dr. A. McLane Hamilton, Dr. D. B. St. John Roosa, and Dr. E. G. Loring, of New York; and Dr. Swan M. Burnett, of Washington, D. C.

The Treasurer's report was read and approved.

CANDIDATES FOR MEMBERSHIP.

Dr. William A. Hammond nominated Dr. W. J. Morton, of New York; and Dr. E. C. Seguin nominated Dr. R. W. Amidon, of New York, for membership in the Association.

The essays of the candidates were referred to the Council for examination and report.

The following amendment, proposed at the last annual meeting, was unanimously adopted:

"ART. VIII. All matters of business shall be attended to by the Council subject to the revision and approval of the Association."

After a discussion by Drs. Mason, Hammond, Gray and others, the following amendment to Art. 8 of the By-Laws was proposed:

"8. Any member of the Association absenting himself from two consecutive meetings of the Association shall cease to be a member, unless what the Council may deem a suitable excuse shall be presented."

GUNSHOT WOUND OF THE NECK — PARALYSIS OF THE CERVICAL SYMPATHETIC, FOLLOWED BY INSANITY,

was the title of a paper sent by Dr. H. M. Bannister, of Chicago, Ill., and read by the Secretary.

The peculiarity of the case reported was, that it exhibited an apparent consequence of these injuries which had not been heretofore reported. The ball from a revolver entered upon the left side of the neck between two and three inches above the clavicular origin of the sterno-cleido-mastoid muscle, directly upon its anterior or inner margin, and, passing backward and a little downward, was lost in the tissues.

The leading symptoms were exophthalmos of the right eye or enophthalmos of the left eye; inequality of the pupils, the right being the largest; and a difference of 1° C. in the temperature of the two sides of the face. There was no tenderness upon pressure on any part of the neck, no ptosis, and no history of any lesion of important nerves save the sympathetic. It was as nearly a case of uncomplicated injury to the cervical sympathetic as he had seen recorded. The chief feature of interest was the connection the case had with mental disorder. There was no history of special neurotic tendency or symptoms of mental disorder antedating the reception of the wound, and no known cause of the insanity, except the wound. He had delusions, hallucinations, and occasionally signs of paralysis. The patient believed himself to be the bishop of Chicago, and thought he had power to make people tell their secrets.

Dr. William A. Hammond, of New York, remarked that Dr. Bannister had barely glanced at the most important part of his case. He had alluded to the fact that the man had *délire des grandeurs*, but had avoided altogether association of disease of the sympathetic nerve with the general paralysis of the insane. He was astonished that so good a scholar as Dr. Bannister should pass so lightly over such an important point. Lesion of the sympathetic existed in most, if not all cases of general paralysis of the insane. In this case, whatever brain lesion there was must have been secondary to the injury of the sympathetic, and, therefore, possibly originated in the latter. The case also went to confirm the opinion that the mind, whatever it was, existed in grey nervous matter wherever found, as well as in the brain itself.

Dr. E. C. Seguin, of New York, remarked that before accepting the case as one of general paralysis of the insane, it would be desirable to scan the semeiology. The only symptom alluded to in the report that would lead to that supposition was the *délire des grandeurs*. Dr. Bannister had not mentioned fibrillary movements of the muscles, peculiar speech, epileptiform attacks, or other varieties of ambitious notions, and simply stated that at one time the patient supposed he was bishop of Chicago, and that he also had power to make people reveal their secrets. But, leaving aside the psychical symptoms, Dr. Bannister had not mentioned the physical symptoms, which were never absent in general paralysis of the insane. He could admit that there was chronic cerebral hyperæmia simply, but should be reluctant to classify the case with general paralysis of the insane.

Dr. Hammond remarked, he was the first to call attention to the fact that, in general paralysis of the insane, physical symptoms manifested themselves before mental symptoms were developed. He had been told by a number of superintendents of insane asylums that they had never, or rarely, if ever, at least, seen a case of general paralysis of the insane in its earliest stages. Consequently, those who had written upon the subject really knew nothing about the primary symptoms, which were physical.

Dr. Putnam, of Boston, objected to the admission of the evidence regarding the patient's psychical condition, and could not see why this injury, situated in an important nerve tract, should act in any other way than through peripheral disorders.

Dr. J. J. Putnam showed a convenient form of German-silver wire *rheostat*, of which the chief merit was that it was cheaper than most instruments of this kind, for the reason that the resistances of the coils had been determined only roughly. Dr. P. claimed that great accuracy is utterly superfluous in such apparatus for clinical work, especially when suitable galvanometers (with a scale indicating absolute quantities of electricity, in Weber's) are used to control the strength of the current.

Dr. Putnam also spoke briefly of the relative advantages of *high and low tension batteries* for clinical work. It had been claimed by a recent writer that the current from the former makes its way more directly through the tissues, between the electrodes, than that from the latter. Experiment, as well as theory, shows this to be an error. It is only when "disruptive" currents are used that the passage of electricity through any conductor fails to obey Ohm's law. The only advantage of high tension batteries (large number of cells, of high resistance) over those of low tension, lies, firstly, in the fact that it is easier to make them durable and constant; secondly, that the strength of the current is affected to a far less degree, with them, by the drying of the electrodes during an application, or by the interposition of a thicker layer of epidermis, or by diminishing the size of the electrodes. This makes it easier to compare the strength of applications made on different days, or to different parts of the body, etc. For ordinary clinical work, however, it is on the whole better to use a battery of which the internal resistance does not greatly exceed that of the human body.

It is then possible to enlarge the electrodes to a convenient size, with the effect of increasing the total quantity of electricity passing, without diminishing its density at any given point; whereas, with high tension and high resistance batteries, an enlargement of the electrodes does not materially increase the total quantity of electricity passing, but does diffuse it through a broader surface of skin, thus diminishing its local action, as for example on a special nerve trunk.

Dr. Hammond, of New York, remarked that rheostats were really of no value whatever in the practice of medicine. The only practical apparatus was something which enabled us to bring a greater or smaller number of cells into the circuit at the same time, and then some temporary arrangement for making and breaking the circuit.

Dr. Putnam allowed all that Dr. Hammond said was true; but the apparatus presented simply permitted of gradual increase in the intensity of the current, and small batteries lasted longer when all the cells were used than when only a part were employed.

Dr. Beard, of New York, remarked that at one time he experimented with all forms of rheostats, but after a while he got out of the way of using any. He thought that his experience was not at all exceptional. Theoretically, they sounded well, and appeared well when written out in full detail; but his experience had been that they possessed no special practical value. For him, pressure of the sponge made a very good rheostat.

Dr. Rockwell.—The method alluded to by Dr. Beard, as practically a good substitute for the rheostat, of increasing or decreasing the pressure upon the sponge electrode, answers very well in many cases where no especial delicacy of manipulation is called for. In other cases, however, it would be an exceedingly slipshod method of procedure, and by it we should certainly fail to obtain the best results possible. In many irritable conditions it is extremely important that the current should be absolutely without break or variation, and these cannot in the strictest sense be secured without the aid of some form of rheostat. Most galvanic batteries that are now constructed, have an arrangement by which it is claimed that the number of working elements may be increased without interrupting the current. My own experience will not substantiate this statement. Even with the greatest precision of manipulation, breaks are apt to occur when least expected or desired, and it is far safer, therefore, to be always provided with some form of rheostat.

Dr. Miles, of Baltimore, remarked that he used the water rheostat constantly, and found it a very great convenience, especially when using electricity on children.

Dr. Seguin remarked that with any of the various sliding bars or dial-shaped current selectors on galvanic batteries it was next to impossible to avoid shocks to the patient, especially the very annoying opening shocks at the close of an application. None of these apparatuses worked exactly in his experience. He thought the chief advantage of a rheostat was the ability it gave to commence with a large number of cells, and gradually increase the current until sufficient sensation was produced—the sensation of the patient being the guide. An objection to the water rheostat was, that while it answered very well up to a certain point, where the metallic points were within a short distance of each other, it was almost impossible to bring them nearer without bringing them together, and thus give rise to a shock. The attachment of a micrometer screw and guard would be advantageous.

Dr. Mason.—I have never used the instrument in therapeutics except on the eye and ear at the N. Y. Infirmary. The whole matter of electro-therapeutics is, and will probably for a long time remain, a physiological one. Hence the importance of all new acquisitions like Dr. Putnam's instrument. For obtaining very mild currents of course it is superior to the water rheostat, when attached as an accessory circuit.

The Association then adjourned, to meet at 8.30 P. M.

FIRST DAY.—EVENING SESSION.

Present: Drs. Beard, Edes, Gibney, Kinnicutt, Mason, Miles, Putnam, Rockwell, Seguin and Spitzka.

The Society being called to order by the President, a paper was read by Dr. George M. Beard, of New York, on

MORBID FEAR AS A SYMPTOM OF NERVOUS DISEASE.

The emotion of fear might be a normal one, varying in degree and kind with race, sex, and individual. It might also be a truly pathological symptom, evidencing in such case disorder of the nervous system. Normal and morbid fears might shade into each other, so that it was difficult to draw the line between them.

These morbid fears might be classified and named for convenience of study, and for the purpose of preventing their confusion with hysteria, hypochondria, dyspepsia, and actual insanity, to which conditions they were often referred. There were quite a number of morbid fears associated with neurasthenia without delusions or hallucinations.

The following classification might be given:

Astraphobia (first described by Dr. Beard a number of years ago), in which there was great fear of lightning during and before thunder-storms, that fear being preceded and accompanied by headache, numbness, pain in the back, nausea, vomiting, and even convulsions. In one of his patients suffering from that symptom, the condition was inherited from the grandmother.

Agoraphobia was a name given by Westphal, of Germany, to those having a fear of places. The term was not an accurate one, and *topo*phobia should be used in its stead, agoraphobia being only a species of topophobia, and embracing those cases in which there was a fear of open squares. As an illustration of topophobia, one of his patients could walk up Broadway without fear or difficulty, on account of the numerous stores and side-streets, into which he knew he could run, in case of supposed danger presenting itself; but he could not walk up Fifth Avenue; he could not visit the country at all. He was a tall, vigorous man, and had no other symptoms of cerebral disturbance. The cases often took an opposite phase: the patients were unable to go to certain places; perhaps where they were first attacked with the evil symptoms. Some found it difficult to go on long journeys. A patient in a distant city wrote that he was coming to visit him, but after getting part of the way he had to return. Some persons had a fear of confined places. A gentleman suffering thus was obliged to camp out in the summer and sleep in a very large room in winter. He finally had to leave the city and become a farmer.

Anthropophobia, that Dr. Beard had lately described, had for its symptoms an aversion to society; fear of seeing any one; sometimes there was fear of women alone; sometimes, as in one of his patients, fear of respectable women alone.

Pathophobia, or fear of disease, was generally known under the name of hypochondriasis. Photophobia was a term that might be applied to some patients who feared that they might fail if they made an attempt to do anything.

Mysophobia, or fear of contamination, had lately been described by Dr. Hammond.

In regard to all these different forms of fear, the following general propositions were made:

1st. They were symptomatic of functional, rarely or never of organic, disease. They were not, according to his experience, found in insanity or sclerotic conditions, and were no form of that disease. The morbid fears of the insane were delusions or hallucinations.

2d. The symptoms might come and go suddenly.

3d. They often were the result of disorders of the reproductive organs in male and female, but might continue long after such disorder had been cured.

4th. They rarely existed alone, but almost always were associated with other symptoms of disorder of the spinal cord or brain, especially of the brain. Among those symptoms were palmar hyperidrosis, dizziness, numbness, shooting pains in the extremities, oxaluria, etc. All those symptoms were by no means uniformly present. The patients might be of great muscular development, and capable of great bodily toil. The treatment required both constitutional and local measures. The former included the whole array of sedatives and tonics; electricity, bromides, counter-irritation at the back of the neck, and counter-irritation of the bowels by means of cathartics. The local troubles should be appropriately treated. The cases could be permanently cured, but not in any short time.

Dr. J. J. Putnam, of Boston, asked if the vertigo ever occurred without being excited or accompanied by any motor idea or intention.

Dr. Beard thought that it did.

Dr. J. J. Mason, of New York, referred to the very common occurrence of fear of lightning and storms, especially with women, and asked why this should be distinguished as a special morbid symptom.

Dr. Beard said that in the cases given there was much more than ordinary fear. The patients often suffered from nausea, vomiting, dizziness, and even convulsions.

Dr. A. D. Rockwell, of New York, said that, without being able to give any special examples to the contrary, he nevertheless could not accept the statement that these fears were always the result of functional, rather than organic derangement.

Dr. Beard, in reply, said that it was his experience that the facts were as he stated.

Dr. E. C. Seguin, of New York, remarked that while the minute study of a symptom of this class was very interesting, it was hardly profitable in practice. He had always found numerous other symptoms with those described by Dr. Beard, and those others were generally of the greater importance. It was better also to look at the symptoms as a whole; to study the patient as a physiological unit, rather than investigate some particular symptom, which might be more curious than important. His experience agreed with Dr. Beard's in regard to the existence of functional derangement only in connection with these morbid fears.

Dr. Beard, in reply, said that he thought the great aim should be to discover the true pathological state of the patient. A study of the symptoms about which he had written helped very often in this direction, and was therefore valuable for diagnosis, prognosis, and treatment. These morbid fears were symptomatic of cerebrasthenia (brain exhaustion), and in a large proportion of the cases (but not in all) that cerebrasthenia originated in the genital organs—uterine displacement and disorders of the neck of the uterus—and diseases of the prostatic urethra in the male, from natural or unnatural excess.

Dr. Webster, of New York, by permission of the Society, related a case of morbid fear that had come under his notice. A woman 40 years old, had always from infancy been afraid of severe storms, high winds, thunder and lightning, and to those who were acquainted with her it seemed that she was insane at such times. When a storm arose at night, for instance, she would rouse the whole house and walk the floor in the greatest distress until it subsided. She did not suffer any pain, but only from nausea. He was told that her mother, while pregnant with her, had been severely frightened by a thunder-storm. The patient's condition was attributed by the friends to that fact.

Dr. Kinnicutt agreed with Drs. Beard and Seguin that morbid fears were evidences of only functional disturbance. In his experience they were very often associated with lithuria or oxaluria.

Dr. Spitzka.—I hold that the position of Dr. Rockwell is sound! Whatever relation fears, more or less akin to those observed as natural manifestations in normal persons, may have to functional derangement, they are certainly highly characteristic of many organic cerebral affections. If Dr. Beard had frequently gone through the wards of an insane asylum, he would not have made the statement that morbid fears are never essential features of insanity. The Germans have a special term (*Kleinlichkeitswahn*) to designate a condition characterized by morbid fears and delusion of powerlessness; as it were, an antithesis to delusions of grandeur and power. The forms of fear described by Dr. Beard, are some of them of such a character that we would be justified in considering the subjects of them as being on the border-land of insanity. I have now under observation two cases of the fear of defilement, recently described by Dr. Hammond; they are both in one family, and I expect to show you a brother of them to-morrow, on which occasion the family history will be discussed. Suffice it to say both these subjects are epileptic.

Dr. Spitzka then related the following case of

REMARKABLE SYMPATHETIC DISTURBANCE.

I regret not having been present when the interesting case of Dr. Bannister was related, as I have seen an instance of mental alienation in association with a sympathetic tropho-neurosis several years ago myself. While visiting the asylum at Ward's Island, in 1876, the year in which I made the autopsies there, I observed a young man of twenty years of age, whose hair on one side was quite grey, while there were sharply marked and intensely colored yellowish brown patches on the same half of his face. On directing Dr. Kiernan's attention to the case he collected for me the following data in regard to him: The patient had been an habitual masturbator for many years. On one occasion, while indulging in his propensity he heard a loud sound as if a pistol shot had gone off in his head, and on the *left side.* He then felt hot on that side, according to his story, and three weeks or a month later, a large carbuncle, as he termed it, opened on the left side of his neck. He was transferred to the asylum in a state of deep depression, and his left pupil was observed to be contracted at the time by Dr. Kiernan. He improved somewhat as to his melancholy, and developed an excessive self-esteem, more, however, in the way of the ostentatiously displayed self-importance of pubescent insanity than of delusions of grandeur. With this the patient was occasionally but mildly excited. The cicatrix of the "carbuncle" is situated on the left side and a little behind the third cervical vertebra. When I saw him there was decidedly an asymmetry of the face, the left side being atrophic; but I observed nothing further about the pupil than that the left one moved more sluggishly than the right. He was at this time very shy and hypochondriacal, especially as to the condition of his hair, which remained grey for two years, although less so at the end of that time. He had spells of taciturnity, but at other times was in a fair mental condition, and at his own request was put at some clerical work.

I am informed that at the discussion the relation of progressive paralytic insanity to lesions of the sympathetic was referred to. Now, although there is good authority for such a connection, and although I myself believe paralytic insanity to be associated with marked vaso-motor disorder, yet, for the reason that the cerebral vaso-motor centre is after all encephalic, and further, as injuries and degenerations of the sympathetic are only exceptionally followed by insanity of any form (never to my knowledge by paresis), I consider the inference faulty. The researches of Poincaré and Bonnet, supporting that theory, have not been confirmed, and as to my own researches on the pathology of paresis, I have found the degeneration of the sympathetic ganglion present only when the cord was intensely degenerated. I have found it absent where the cord was slightly or not all involved. No importance can be attached to the lesion of the sympathetic therefore, the essential lesion is cerebral.

In other cases of gun-shot injury of the sympathetic, notably in one reported by an Austrian surgeon during the campaign of 1866, no mental symptoms whatever have been observed.

In a short discussion which followed, Dr. Spitzka referred to the fact that experiments and investigations showed the sympathetic system to be independent of mental disturbances, and that it could be greatly injured or diseased, and yet the mind not be at all affected.

The Society then adjourned to meet Thursday, at 2.30 p. m.

THURSDAY, JUNE 19TH.—SECOND DAY—AFTERNOON SESSION.

The Association was called to order at 2.30 p. m. by the President.

Present—Drs. Beard, Edes, Gray, Hammond, Kinnicutt, Mason, Miles, Morton, Putnam, Rockwell, Schmidt, Seguin, Shaw, Spitzka, and Van Bibber.

The minutes of the previous day were read and approved.

The Council reported that it had examined the papers presented by the two candidates for membership, and recommended that they be elected members by the Association.

On motion, the By-Laws were suspended, and the Secretary was then instructed, by motion, to cast an affirmative ballot for Drs. W. J. Morton and R. W. Amidon, of New York.

The amendment to the By-Laws, offered yesterday by Dr. Gray, was adopted by the Association.

Dr. Hammond offered an additional amendment, as follows: "The presentation of a written neurological communication shall be deemed a valid excuse for absence from an annual meeting of the Association." Adopted.

The President announced the following Committee on Nominations: Dr. H. D. Schmidt, of New Orleans; Dr. E. C. Spitzka, of New York; Dr. A. D. Rockwell, of New York; Dr. J. J. Putnam, of Boston; and Dr. J. C. Shaw, of Brooklyn.

Dr. Rockwell presented two cases: I present these two cases because they illustrate two very important points in therapeutics: 1st, The necessity for, and the good results that come from perseverance in seemingly hopeless cases of infantile paralysis. 2d, A suggestion in regard to the differential indications for the use of galvanism and faradism in the relief of pain. This little boy, aged four, sent to me by Dr. McElroy, of Mott Haven, suffered in 1877 from a severe attack of chills and fever, followed by convulsive seizures. Complete paralysis followed, and six months subsequently, after the leg had become cold and atrophied to the last degree, with entire loss of both farado- and galvano-muscular contractility, I first saw the child. It was not until after six weeks of treatment that there was the slightest evidence of returning galvano-muscular contractility, and six months of persistent effort followed before there was any response to the faradic current. At the present time the contractions are good—the limb warm and nearly as large as its fellow, and, with the aid of a chair, the little one can move around quite readily.

In consideration of the absolute and long-continued paralysis and loss of electric response to either current, the results have impressed me deeply, and should teach that no case of infantile paralysis should be hastily abandoned to its fate, even though it seem possible, as in this case, that commencing fatty degeneration existed.

The second point is illustrated in the case of this gentleman, sent to me by Dr. Malcolm McLean, of Harlem: Last Christmas, without knowledge of special exposure, but subsequent to much mental depression for cause, the patient began to suffer from extreme pain in the back and along the sciatic nerve. The pain was dull and aching, with occasional sharp twinges. At times he was quite helpless. He was treated in many ways—even by cauterization—without experiencing any relief, and latterly he has been worse. I first saw the patient two weeks ago (six months since the first attack) and although well aware of the probable indications for treatment, but in order to test the case thoroughly, I made a careful labile application of the galvanic current, which served to increase the pain. Two days after I resorted to faradization, placing the sound as well as the lame foot on the negative pole, using an exceedingly fine current, carefully avoiding all motor points and carefully refraining from producing appreciable muscular contractions.

The patient has been thus treated but three times, and as you will observe, is nearly well. The points of this case that I wish particularly to emphasize, are first, that in differentiating between the use of the two currents for the relief of pain, the effects of pressure are most useful guides, and that while true neuralgia, as defined by Anstie, and pain generally yield as a rule more readily to the galvanic than to the faradic current, the latter is in some cases, not only invaluable, but relieves, where galvanism is worse than useless, since it serves only to exaggerate the existing distress. This case is a good example of this statement, for on my first examination, I found that firm and prolonged pressure did not increase the pain, but afforded relief. Slight pressure, on the contrary, caused, all along the outer aspect of the thigh, very intense pain. Hysterical neuralgias, and the so-called pseudo-neuralgias, which are simply forms of pain, occupying certain areas, and running seemingly in the direction of certain nerves, undoubtedly indicate the faradic current. In these cases it is well known that firm pressure tends to relieve, while light touches aggravate the distress.

With reference to the case of infantile paralysis, Dr. Miles asked if voluntary movements had returned to the muscles.

Dr. Rockwell replied that they had; that they were marked, and that they had returned after an absence of six months, after all normal recovery had taken place.

Dr. Miles remarked that he had succeeded in such cases in restoring galvano-muscular contractility, but not voluntary action.

Dr. L. C. Gray, of Brooklyn, asked which pole was used for the relief of pain.

Dr. Rockwell replied that he used the descending current.

Dr. Hammond remarked that he had reached the conclusion that it did not make any difference with regard to which pole was employed, and that the one was as good as the other.

Dr. Rockwell remarked that to reach correct conclusions upon this, as upon other interesting points, considerable observation was necessary. He had seen cases in which, after obtaining very good results from the use of the descending current, he had, in order to hasten the improvement, used the ascending current, and all the pain returned.

Dr. Hammond remarked that he had seen such a result from the continuous use of one current, either the ascending or descending.

Dr. Gray remarked that his experience had led him to the conclusion that it made but little difference which pole was employed, except about the eye or face. Upon the face he had found a difference in the effect produced by using different poles, and he had explained the difference by the fact that in physiological experiments and therapeutical uses of electricity the conditions were essentially different; in the one the nerve was laid bare, while in the other it was more or less deeply covered with tissues. When deeply covered, the electricity became so diffused that its direct action upon the nerve was questionable, while in regions in which the nerves were less deeply covered the conditions present in physiological experiments were more closely approximated.

Dr. Gray then referred to a case of tic douloureaux in which galvanism did good for the first day, and for the next two or three days it did harm. On the *first* day he used the descending current, or positive pole; and in the succeeding two or three days he employed the ascending current or negative pole. He at once returned to the use of the descending current, and so long as it was continued there was a great amelioration of symptoms.

Dr. Beard remarked that the most interesting feature of Dr. Rockwell's case was the fact that the faradic current gave relief to the pain. It had been stated for such forms of disease we must use the galvanic current exclusively. In the cases of sciatica reported by Dr. Gibney, of New York, that idea was involved, and it was a popular opinion in Europe at the present time; but it was erroneous.

The suggestion made by Dr. Rockwell with reference to pressure as an indicator in differentiation as to the use of galvanism or faradism to relieve pain, was perhaps a good general guide; but it was not applicable to all cases.

With regard to the direction of the current he thought it was one of the most difficult things in the world to differentiate, so that we could eliminate any source of error. For the present he had abandoned it. Theoretically there should be a difference in the action of the poles, and difference in direction of the current with regard to the poles; but practically, the elements of error were so that he confessed he was not always able to make the differentiation.

Dr. E. C. Seguin asked Dr. Beard if he recognized the polar method in distinguishing electrical reaction. Dr. Beard replied that he did.

Dr. Seguin thought that settled the question, for by that method decided differences could be distinguished. If we proceeded to test the patient according to Erb's instructions, we obtained decided polar reactions and peculiarities, and those peculiarities which were normal were imitated in pathological conditions, and could be recognized in the first examination of a case of paralysis. With regard to relief of pain, his experience had been favorable to the sedative effects of the anode, provided a mild current was used, and to the exciting effect of the cathode.

Dr. Beard remarked that in a majority of cases pain was relieved when a sufficiently mild current was used. For the first four or five years of his practice he always knew what pole he employed, but of late years, in very many cases, he did *not* know, and he was not able to recognize any change in the results of his electrical treatment.

Dr. Putnam remarked, it was proper to say that perhaps we invoked the aid of physiological experiments too much, inasmuch as those effects were suddenly produced, and passed away as quickly.

Dr. W. J. Morton, of New York, remarked, he felt quite sure that, in neuralgia of the trigeminus the positive pole reduced pain in cases in which the negative pole produced no marked effect. He thought that the determination of the polar difference should not be lost sight of in electro-therapeutic treatment, and that the very fact of the existence of such a difference implied a corresponding difference in the therapeutic effect of the two currents. Nothing was better established than the peculiar catalytic action of the two poles upon the human tissues, at least in certain cases; the positive pole of a body battery applied to the open surface of an indolent ulcer would cause it to granulate and heal in a few days, while the negative, applied at the same time, on the sound parts, produced a destruction of tissue in the shape of small pustules, which would become ulcers. He had been accustomed to consider the positive pole as consolidating, organizing and sedative to the part applied, and the negative as liquefying, disorganizing and destructive; this, at least, he felt sure was the effect in the treatment of ulcers by galvanic electricity, and there was no apparent reason why the same catalytic effect should not hold true of the electrodes of the galvanic battery whenever applied to any tissue. It was fair to presume that the special effect inherent in the nature of the poles should extend itself to a certain extent in the line of the tissue circuit connecting the two; and on the ground also of the catalytic difference in the two poles, he should be unwilling to think that it made no difference in which direction the current was applied.

Dr. Gray thought there could be no question but that there was a physiological difference between the poles, but he failed to see that there was convincing evidence to prove any therapeutical difference. He then referred to a book published last year by L. J. Teissier, of Paris, in which the author had manifestly taken considerable trouble to collect evidence with reference to the therapeutical value of electricity, but in looking through it he had failed to see any convincing evidence to prove any therapeutical distinction between the poles. He (Dr. G.) did not say there was no difference, but he simply believed that it had not been proven.

Dr. Hammond thought Dr. Gray would change his opinion when he witnessed the result of electrical treatment of ulcers.

Dr. Gray remarked that he had used electricity in the treatment of abscess, and that he had not seen any difference in the results obtained by the use of different poles.

Dr. Hammond remarked that he knew there was a therapeutical difference between the positive and the negative poles, and that fact he first established to his own satisfaction in a series of cases of ulcers treated by galvanism at the Baltimore Infirmary several years ago. He knew that a silver (*positive*) plate placed upon an indolent ulcer, with a zinc plate (negative) above facilitated healing; whereas, applied in the opposite manner, the condition was invariably aggravated. The single pair of plates gave the least possible intensity.

Dr. Gray remarked that he did not question the difference in the results in the treatment of ulcers; but he should not, from that fact, argue with regard to the therapeutical value upon the unbroken skin.

Dr. Hammond remarked that he was aware it required a great deal of experience to settle some of these difficult questions in electro-therapeutics. In his opinion it did not make any difference which current was used to give relief to pain. We already knew that there was a physiological and a chemical difference between the action of the poles, and the question then was whether there was any therapeutical difference, and he thought there was.

Dr. Beard thought there was no chance for dispute with regard to the effects produced upon ulcers by different poles, the positive being the more efficacious for healing purposes. He believed there was one thing sure—namely, that in all countries where electricity was used, there was less and less tendency to insist upon polar use. The best writers took the view that the practical difference between the positive and negative pole was not so much as formerly supposed.

Dr. Gray remarked that he did not believe there was any difference with regard to the direction of the current; and that if there was a difference it was between the poles.

Dr. Miles remarked that he had seen unmistakable difference between the two poles in allaying pain. In the treatment of myalgia occurring in his own person, he had found that the positive pole gave relief much sooner than the negative.

INFANTILE ENCEPHALITIS FOLLOWED BY ATHETOTIC SYMPTOMS.

Dr. E. C. Spitzka exhibited a patient who had athetotic symptoms, as he believed, the result of infantile encephalitis.

GLOSSO-LABIO-LARYNGEAL PARALYSIS.

Dr. Hammond presented a patient suffering from the above disease. The treatment was, phosphide of zinc, one-tenth of a grain, t. i. d., and extract of nux vomica, one-third of a grain, t. i. d. Electricity was also used; and, while the patient was certain that he was improving, Dr. Hammond thought his condition would never be substantially improved.

MYELITIS WITH FORMATION OF CAVITIES OR VACUOLES IN GANGLION CELLS IN ANTERIOR HORNS OF SPINAL CORD.

Dr. R. T. Edes, of Boston, read a paper in which was given the clinical history of a case that developed, orderly and symmetrically, symptoms referable to the spinal cord. There was symmetrical atrophy of the legs below the knees, and the arms below the elbows, and the left pupil was larger than the right. The case was of a little less than four months' duration, and on microscopic examination of the cord the white substance was found normal in every respect. In the grey matter the only change consisted in the presence of large polygonal spaces or vacuoles in varying numbers in the anterior horns. The processes of the ganglion cells seemed slightly changed in places—shrunken and shortened. The lesion was found in the cervical enlargement, in the dorsal region, and in the lumbar enlargement. The specimens seemed to illustrate that parenchymatous changes might take place independent of blood-vessels or neuroglia.

Dr. Putnam remarked that he had seen a case closely resembling Dr. Edes' in clinical history, except that the fever was higher, the temperature rising as high as 104°–105° F. There was the same progressive paralysis, and, on microscopical examination, spots of softening were found in the lenticular ganglion upon the left side. In the cervical region there was an evident change in the ganglion cells, consisting in large collections of fat. He thought that it was possible, if the case had lasted longer, the change might have gone on to the formation of vacuoles.

Dr. E. C. Seguin referred to specimens in his possession, of acute myelitis, in which the symptoms of transverse and total myelitis were developed within twenty-four hours. Death occurred at the end of the sixth or seventh week, and on examination he found extensive softening of the lower dorsal cord, which microscopically exhibited no special lesion, but in sections below, the first of moderate worth, there were found vacuoles, such as described by Dr. Edes.

He thought the clinical history of Dr. Edes' case belonged to poliomyelitis more nearly than anything else.

Dr. Edes remarked, that although his attention was for the first time called to the change present in his specimens, he looked through many sections of other spinal cords which he had, and in a single section he found one cell of the kind described, and in a case which was recorded as healthy. He now thought it was a cord not above suspicion.

Dr. H. D. Schmidt, of New Orleans, remarked that he had observed vacuoles in the colored blood corpuscles, and the only way in which he could explain their presence was that a portion of the protoplasm had decomposed. He had frequently seen the same appearance in the sympathetic system, between the ganglionic body and the protoplasmic portion of the processes. In that case he had ascribed it to contraction of the protoplasm and filling of the space thus produced between the enclosing membrane and the body by some liquid or gas. Dr. Schmidt thought that the vacuoles seen in Dr. Edes' specimens were fat globules; for, in some of the ganglionic cells there could be seen granular degeneration, which needed to be only a little more marked to become fatty infiltration.

Dr. Spitzka regarded the use of alcohol as prejudicial against the proper preservation of nerve tissue, in that it favored the formation of crystals of leucin and other like substances. Even where chromic acid was used, he had found little spore-like bodies, which presented the appearance of myelin, but which were not myelin.

Dr. Edes remarked that he had compared his specimens with many others made in the same manner, and did not think the method of preparation and preservation of the tissue had anything to do with the change exhibited. Furthermore, the symmetrical distribution of the lesion, together with the symmetrical character of the symptoms pointed to a very close relationship with each other.

Dr. Schmidt did not think they were artificial, because there were but few cells involved; there were many cells around them which were not at all affected.

Dr. Spitzka.—While I do not mean to intimate that the lesions in Dr. Edes' case are anything but genuine, I consider it unfortunate in any case of disease involving the nerve centres, to use alcohol, as it produces many curious appearances, even in the freshest and healthiest brains. I should like to ask if the cranial nerve nuclei were examined, and whether any lesions were found in them?

Dr. Edes.—They were examined and no lesions found.

Dr. Spitzka.—Then this would be one of those cases in which, on the borders of an actual lesion, the lesions one would expect to find, as in this case, for example, those determining the facial symptoms, were impalpable?

Dr. J. C. Shaw.—In examining these specimens, I see that there is a perceptible sclerosis of the lateral columns. This is of interest to me, as I have recently published a case of progressive muscular atrophy with bulbar paralysis, in which there was myelitis of the anterior horns with sclerosis of the lateral columns, but without contraction. Now this case of Dr. Edes, it appears to me, has some bearing upon the cases described by Prof. Charcot as lateral amyotrophic sclerosis, and it would appear that sclerosis of the lateral columns due to myelitis of the anterior horns is not an uncommon condition. This case is of double interest, as it has lasted only six weeks.

Dr. Spitzka.—There certainly is lateral sclerosis of a limited extent here.

The Secretary read a communication from Dr. J. S. Jewell, of Chicago, expressing his regret at not being able to be present because of serious sickness in his family.

Dr. J. J. Mason exhibited micro-photographs illustrating

THE HISTOLOGY OF THE MEDULLA OF THE ALLIGATOR.

Two years ago I had the pleasure of exhibiting to this Association sections of various portions of the central nervous system of the alligator. During the winter of 1877 and '78, following, a residence of several months in Florida enabled me to continue the study of the subject, with abundant material. It was then my purpose to prepare an exhaustive treatise; but, before the text could be written, the first part of Rabl-Rückhart's article appeared in the *Zeitschrift fuer Zoologie*, January, 1878.

His researches had their source in the accidental and, to me, untimely death of five large Mississippi alligators at the Berlin aquarium in the winter of 1875 and '76. So far, he has not published any of his microscopic work, all of it being reserved for part II.

While, therefore, we can no longer enjoy a monopoly in the study of the nervous system of this species of American reptiles, let us hope that the audacity of our transatlantic colleague, in thus superseding us on our own ground, may tend to awaken a larger degree of patriotism in our scientific work.

I wish, to day, to call the attention of the Association to some peculiarities of structure which are to be found in the medulla oblongata and spinal cord of this alligator, illustrated by sections and photo-micrographs, and to show a few sections of the medulla oblongata of the skink lizard (*Scincus quinquelineatus*, Holbrook), the gopher turtle (*Testudo polyphemus*) and the bullfrog (*Rana pipiens*). At our next meeting I hope to be able to present a more complete collection of photographs, illustrating as fully as possible all the most important features of the subject. The photographs will be permanent, printed by the new process of Bierstadt, called the artotype.

The sections on the table show, first, a group of remarkably large nucleated multipolar cells situated within the septum of the medulla oblongata. These bodies send out long processes; and, in one of the sections, a process appears to enter another septum at a considerable distance from the first. They are found chiefly in sections made just behind the point of exit of the acoustic nerves. Their size is often double that of other cells in the medulla, and of any that I have yet seen in the spinal cord. They are found in even larger numbers—though of relatively smaller size—in the same region of the medulla of the skink, extending well towards the cervical region, but are absent (as a group, at least) in the medulla of land turtles and frogs. Stieda does not mention nor figure them in his work on the structure of the nervous system of the turtle (*Testudo græca*), but in the same monograph, part I., on the nervous system of the axolotl, page 17, says: " In the spinal cord a cell-grouping exists on either side —especially in the inferior horn; in the median line no cells are to be noticed; here in the medulla oblongata nerve cells lie on the whole periphery of the grey matter, and *also* in the *median line.* At about the middle of the medulla oblongata the cells begin to show themselves, and can be followed farther in successive sections. As regards the appearance of the nerve cells in general, they are of the same form and size as in the spinal cord, while in the anterior portion of the medulla oblongata I find—especially in the median line, or in its immediate vicinity—remarkably large cells, with long processes.” These cells may, and probably do, correspond with those which I have found in the alligator and skink, for they were found near the acoustic nerve, although the body of the cell is represented in the cut as lying outside, while its processes alone are within the septum. As shown by my photograph, in the alligator, the cell body itself is imbedded in the muscles of the septum.

Second : An arrangement of spindle-shaped cells in the inferior horns of both the cervical and lumbar enlargements of the spinal cord. This arrangement relates to the processes which run parallel with each other towards the superior horn, describing a series of curves with their convexities towards the central canal. It is also seen in the turtle, first, I believe, by Lockhart Clark.

Third: The thickness of the cord of the alligator, in different regions, is well shown by photographs 3 to 6, inclusive.

The negatives were taken through the same objective, so that we have only to measure the photographs to obtain a sufficiently correct estimate. These measurements are as follows for cervical and lumbar enlargements:

Cervical—horizontal	$4\frac{1}{16}$	inches.
“ vertical	$2\frac{6}{8}$	“
Lumbar —horizontal	$4\frac{2}{16}$	“
“ vertical	$2\frac{7}{8}$	“

The grey matter of the lumbar enlargement on a cross-section exceeds but slightly in amount that of the cervical enlargement (*when measured*), so that in reality there is little or no difference between the dimensions of the cord in these two regions. Such an equality in thickness does not exist in the cords of animals destitute of a tail, or in those with whom it is merely an appendage. The apex of the inferior horn is, as a rule, broader and more angular in contour in the cervical than in the lumbar enlargement; and this feature, together with the existence here of two distinct groups of cells, as in the cord of the frog, enables us to distinguish under the microscope between two preparations made from the two different regions.

The sections from the cord of the Florida gopher are of interest, as showing the same general conformation of the grey matter as was observed by Stieda in *Testudo græca*, with the exception of the dorsal region, when the inferior horns appear nearly straight.

Dr. Spitzka.—The groups of cells shown by Dr. Mason are peculiar for their size, and I know of no other animal in which the raphe cells reach these dimensions; it is certainly an interesting observation in every way. I have observed cells in the raphe of lesser dimensions; in man, where they occur in large numbers, sometimes in the altitude of the hypoglossal nuclei, as well as in the yellow spotted and snapping turtle, and the Pseudopus. I consider their occurrence in the medulla oblongata as due to a local modification of the embryonic laws of growth, which condition does not obtain in the cord. I hold these cells to be auxiliary to the cranial nerve nuclei.

(In response to Dr. Mason's theory as to their relation to the length of the tail.)

Dr. Spitzka.—I do not think that these cells can have a special relation to the size of the tail; the raphe would be a very unfavorable place for the location of such cells, since its fibres cross the nerve-axis. Furthermore, I do not believe in such distant projection from grey matter of the third category. If these cells be analogous to the similarly situated cells of the chelonia, then a single instance would contradict it. The yellow spotted turtle, with a short, weak tail, has this group twice as large as the snapping turtle, with its tail as muscular almost as the alligator's, and as long in the male as the entire shell.

(Dr. Mason stated that it was only an informal suggestion, and not even a theory with him.)

Dr. Schmidt.—Regarding Dr. Mason's specimens, I asked to what group of ganglion cells he supposed the ganglion cells of the raphe, which he exhibited, belonged, and whether they might not be analogous to the nuclei of the stratum zonale, or be compared with those ganglion cells in the white columns of the spinal marrow of the amphiuma?

The Society adjourned to meet at 8.30 p. m.

SECOND DAY—EVENING SESSION.

The Association was called to order by the President.

Present: Drs. Amidon, Beard, Hammond, Kinnicutt, Mason, Miles, Morton, Rockwell, Seguin, Spitzka and Van Bibber.

The proceedings began with remarks by Dr. Wm. A. Hammond, of New York, on

METALLO-THERAPY.

He referred to its history, spoke of his own work in connection with the subject, and expressed his great surprise that a man of such scientific training and experience as Charcot should have lent himself to so vile a humbug. Dr. Hammond showed disks of various metals which he had used in his experiments, and asserted that the one of tortoise-shell was the most efficacious, and was the one used most.

The subsequent discussion of the matter showed it to be the unanimous feeling of the Society that the claims of metallo-therapy were unfounded and absurd.

In view, however, of the fact that it was not yet universally condemned by physicians, and even had the support of some eminent men, it was voted that a committee be appointed to investigate the matter, both in its medical and psychological phases.

Dr. W. J. Morton, of New York, then read a paper upon the

TOXIC EFFECTS OF TEA.

The subject, he said, was best studied by examining that class of men, such as tea-tasters, who habitually took tea in large amounts. It was, however, not easy to obtain extensive data concerning those men, for they feared if the facts became known it might injure their business. Five cases, however, had been collected, and those, together with experiments performed by the writer upon himself, formed the basis of the paper.

The bad effects of tea-tasting were known and recognized by the tea-tasters themselves, and few could carry on the business many years without breaking down. One tea-taster estimated that he got about half a pound of tea into his system during a day. It has been said that the symptoms from which tea-tasters suffered were due to alcohol or dyspepsia, but the facts collected showed the contrary.

The writer then gave the history of the cases referred to, and of the experiments upon himself.

The following is a *resumé*: First, as to the immediate effects of moderate doses, there was in the cases observed: an elevation of pulse, increase of respiration, agreeable exhilaration of mind and body, a feeling of contentment and placidity, an increase of intellectual and physical vigor, with no noticeable reaction.

The immediate effects of an excessive dose were: rapid elevation of pulse, marked increase of respiration to the extent of about one-third, increase of temperature, no period of exhilaration, but immediate and severe headache, dimness of vision, ringing in the ears, dullness and confusion of ideas. Following that was a severe reaction: exhaustion of mind and body, tremulousness and "nervousness," and dread of impending harm, that could not be relieved by taking more tea.

The effects of continued doses were a continuance of the tremulousness, extreme susceptibility to outside impressions, constipation, diminution of urine, and marked influence on the metamorphosis of tissue as shown by the diminution in the amount of urea. Thus, in the week during which the writer was taking toxic doses of tea, the amount of urine fell from f. ℥ xl. to f. ℥ xxxii. per day; and in the same time the urea fell from gr. 591 to gr. 422 per day. The sulphates, phosphates and chlorides were increased.

The results, as regarded the diminution of urea, agreed with previous experiments, but showed the influence of the tea much more strikingly.

From the study of the drug's action, Dr. Morton arrived at the following conclusions:

1. That with it, as with any other potent drug, there was a proper and an improper use of it.

2. That in moderation it was a mild and pleasant stimulant, followed by no harmful reaction.

3. Its continued and immoderate use led to a very serious

group of symptoms, such as headache, vertigo, ringing in the ears, tremulousness, "nervousness," exhaustion of mind and body, with disinclination to mental and physical exertion, increased and irregular action of the heart, and dyspepsia.

4. The mental symptoms were not to be attributed to dyspepsia.

5. It diminished the amount of urine, and retarded the metamorphosis of tissue.

6. Many of the symptoms of immoderate tea-drinking were such as might occur without a suspicion of the real cause.

The paper being open for discussion, Dr. J. J. Putnam, of Boston, asked whether the symptoms of weakness, nervousness, etc., which followed the taking of large doses, were those of reaction, or were the continued toxic effects of the drug.

Dr. Morton thought they were the latter.

Dr. Hammond referred to a case of his where the patient suffered intensely from neuralgia, brought on, he believed, by tea-drinking.

Dr. Miles asked if the tea-tasters were subject to any great mental strain or anxiety in their business.

Dr. Morton said that they were not. Referring to the influence of alcohol, he asserted positively his belief that it had nothing to do with the train of symptoms he had given. There was, to be sure, very often a craving for alcohol, but it was not uniformly felt, and the danger of drinking was appreciated by the tea-tasters.

The Association then adjourned to meet on Friday at 2:30 p. m.

FRIDAY, JUNE 20TH.—THIRD DAY—AFTERNOON SESSION.

The Society was called to order at 2:30 p. m. by the President.

Present: Drs. Amidon, Beard, Edes, Gray, Hammond, Kinnicutt, Miles, Morton, Rockwell, Seguin, Schmidt, Spitzka, and Van Bibber.

The minutes of the previous day were read and approved.

REPORT OF COMMITTEE ON NOMINATIONS.

The Committee on Nominations made the following report:

For President—F. T. Miles, M.D., of Baltimore, Md.

For Vice-President—R. T. Edes, M.D., of Boston, Mass.

For Secretary and Treasurer—E. C. Seguin, M.D., of New York.

For Councillors—Dr. F. K. Kinnicutt, of New York, and Dr. L. C. Gray, of Brooklyn, N. Y.

An election by ballot was held, and these gentlemen declared duly elected by the President.

Dr. Geo. M. Beard presented two patients who illustrated anthropophobia and topophobia. Both were strong, vigorous men.

THE DOSAGE OF ELECTRICITY.

Dr. Geo. M. Beard read a paper on the above subject, in which he stated that the dosage of electricity was a complex result of a number of different factors.

1. The strength of the current.
2. The length of the application.
3. The quality of the application, and
4. The method of the application.

1. The strength of the current.—It was difficult to estimate that, and to say that we had treated a patient with so many cells was saying but little. The size of the electrodes, the manner of their application, and the moisture of the skin, must all be taken into consideration. The resistance offered by the skin was also an important element, for it was variable, and, besides, the quality and temperature of the water were important considerations in making an estimate.

The amount of electricity passing through the body was varied by—

a. The electro-motive force of the battery.
b. The internal resistance within the battery; and
c. The external resistance outside of the battery.

2. The length of the application could not be determined with minute precision. As a rule, European neurologists made shorter applications than did the American. There was no question but that long applications exhausted muscles, and an application of one or two minutes' duration to a paralyzed muscle was all that was beneficial. The beginning of electrical treatment should be with short applications and mild currents.

No absolute rules could be given regarding strength of current or length of application. Other things being equal, a stronger current required shorter applications. Other things being the same, the galvanic current was not so well borne for a long time as the faradic current of corresponding strength.

3. The quality of the application.—The use of mild currents at first was wise. Experience compelled us to admit that stimulating as well as sedative effects could be obtained by negative as well as positive poles. Practically, the difference between the positive and the negative poles, and between the ascending and the descending currents, was one of degree rather than kind.

4. The method of application was largely included in what had been said. He had long ago recommended the terms, medium, mild and strong currents as the best approximation possible to a fair description of the doses of electricity.

Again, the dosage of electricity was modified by the external position of the poles. The same strength of current with the negative pole was a stronger application of electricity than with the positive pole in the same locality.

Even the best constructed galvanometers interposed in the circuit could not be an accurate measure of the dosage of electricity, for the reason that the amount of electricity passing through the body of the patient would vary with the pressure on the sponge and with the position of the electrodes. Consequently there might, in different applications, be the same number of degrees indicated by the galvanometer, while the applications really were very different in their character and in their effects, because the electrodes were differently placed.

Temperament was another element to be taken into consideration in regulating the dosage of electricity.

Finally, what was true of familiar drugs, and the conditions regulating their dose, was true of electricity.

DISCUSSION.

Dr. A. D. Rockwell—There are probably few remedies to the effect of which there is such a varying degree of susceptibility as to electricity. There is no absolute law by which we can be governed; and, therefore, each case must be studied by itself. Let the first applications be tentative: for until the patient has been submitted to a thorough electrical test, it will be impossible to decide the exact strength of current best suited to the case in hand. My own experience teaches, that where we aim to secure the tonic effects of electricity, or to relieve pain, the applications should never be carried to the point of inducing discomfort.

Very frequently, however, in the treatment of paralyzed muscles, this will not hold, and it becomes necessary to use a current strength that is not at all agreeable. When it becomes necessary to measure the galvanic current with some degree of accuracy, a properly constructed galvanometer is a useful accessory. I do not refer to the ordinary short coil galvanoscopes. These are so delicate that the needle is deflected to 90° under the influence of two or three cells, and are of service only when we wish to know whether a current is present, and if so, what is its direction. I have for a number of years used in my office a galvanometer, constructed at my suggestion, which has answered my purpose admirably well. It is of the long coil variety; is provided with a shunt, giving a resistance equivalent to 150 miles of telegraph wire. A freshly-charged zinc carbon battery deflects the needle about one degree for every cell. As the battery becomes weaker, it becomes necessary to increase the number of cells to deflect the needle a certain number of degrees; this deflection indicates approximately the strength the current cells should be increased. Of course the usual coils were so delicate that they were of no practical value except to show the presence of a current, and determine the presence of the positive and negative pole.

With regard to the direction of the current, he felt able to say from an experience in several thousands of cases, that for the relief of pain, the descending was much to be preferred to the ascending current. Whether it depended upon the direction of the current or upon polar action, he was not prepared to say. In a number of instances of true neuralgia, in which the descending current had relieved, a reversal of the method had again increased the pain.

With reference to general faradization, he could only account for the neglect of that method on the ground of great inconvenience to which the patient was subjected in the application; also the time required, together with the experience actually necessary to its satisfactory use.

Dr. L. C. Gray, of Brooklyn, remarked with regard to the therapeutic effects of general faradization, that he had had several cases which had made him a rather firm believer in its good effects for certain patients. He then referred to a case in which a lady suffered from symptoms upon which only a general diagnosis could be based. She had a feeble pulse, general physical weakness, chronic stomatitis and a variety of symptoms; and nothing which she had tried seemed to do her any good. He resorted to general faradization. After six sittings her stomatitis was cured; her appetite was better than it had been for a long time; her pulse was remarkably stronger, and she was able to walk very well. He had had a number of other cases in which the permanent effects of general faradization had been distinctly marked. The manner in which he applied it, was to have the patient place one foot upon a small sponge, and hold a sponge electrode in the opposite hand, and shifting them from one side of the body to the other. In his cases, that method had been sufficient, and was so simple that it gave no inconvenience to the patient.

Dr. Rockwell protested against the method described by Dr. Gray as a substitute for general faradization, because it would do more damage than good. Nothing partial could be with benefit substituted for general faradization.

Dr. Gray remarked, with reference to Dr. Rockwell's criticism of his method of application, that the "proof of the pudding is in the eating." What patients wished was to be cured, and, of course, it was no matter whether they were cured according to the rules of science or any other way which they chose to accept. The changes for the better were indisputable in his cases, and they were remarkable; and that fact was rather good proof of the lack of injury in the departure from the method formerly advocated.

Dr. Hammond asked Dr. Gray what class of cases he cured by having the patient hold a pole first in one hand and then in the other.

Dr. Gray replied, that they were just such cases as he was now attending, one of which was sent to him by Dr. Hammond. In that case the patient had chronic rheumatism, a morbid, regurgitant murmur, and general debility.

Dr. Hammond.—And spasm of the sterno-cleido mastoid muscle?

Dr. Gray.—Yes; and that is steadily getting better.

Dr. Hammond remarked, that probably the spasm of the muscle would get well of itself. He doubted whether a case in which the disease was limited in its manifestations to spasm of the sterno-cleido mastoid muscle could be cured by changing the electric poles from one hand to the other. If it could be limited in that manner, he could give up general faradization altogether.

Dr. Gray remarked, he did not claim that the electricity was all the treatment which the patient received, for he had used subcutaneous injections of atropia.

Dr. Hammond, in confirmation of Dr. Beard's statement regarding the relative insensibility of the cervix uteri, stated that many years ago he had constructed an instrument with which the cervix could be galvanized for the purpose of establishing menstruation in certain cases. It consisted of a double spoon, the outer part being made of hard rubber, and the inner was metallic. By that instrument the cervix could be grasped between two metallic surfaces, and held firmly by means of a sliding ring. The instrument terminated in two wires, which were connected with the poles of a battery. In all cases where the current was passed through the cervix the patient was absolutely insensible of its passage. The same thing was true not only of the neck of the womb, but of the male urethra. When one pole was placed in the urethra and the other held in the hand, or placed upon the back or upon the pubis, the patient would not feel the internal pole until considerable current was felt in the hand or other part, and then only very slightly. He had another apparatus constructed, which was essentially a catheter that terminated in two metallic disks, so that both poles could be introduced into the urethra. By revolving the instrument, it could be made to touch any part of the urethra, and even then the patient was not sensible of the passage of the current. But that did not hold good with reference to the galvanic current.

Dr. Beard remarked that it made a difference with regard to the size of the electrodes used. With the ordinary sound in the male urethra, a rather strong galvanic current could be used without producing pain, and it would be felt quicker than the faradic current. The general statement he had made was correct. But there was this distinction, that the faradic current was felt more with each portion of a minute than the galvanic current. That was well known by all. It was a fact that tonic and sedative effects of electricity could be obtained in the manner which Dr. Gray had mentioned, but it was not a method that should be recommended. He had no doubt that considerable tonic influence upon the body could be obtained in that manner, but it was absurd to adopt it when it was possible to use any other method.

Dr. Gray asked Dr. Beard what his method of using general faradization was?

Dr. Beard replied that it varied very much indeed. Practically undressing was a minor trouble, and it was the method he usually adopted. In some cases only the shoes and stockings were removed, but it was necessary to raise the clothes so that the electrode could be carried over various parts of the body.

Dr. Gray remarked that the amount of electricity made to pass through the system in that manner was about the same as by his unscientific method. With the sponge under one foot and the electrode in the opposite hand, the current passed through the foot, the limb, traversed the cavity of the body, traversed the tissues of the body, and came out at the palm of the hand, and by reversing the procedure, the current was made to pass through the tissues in an opposite direction. The only parts which were not collaterally affected were the neck and the brain, and they might be affected by allowing the patient to take one electrode in the hand while the other was passed over those regions.

Dr. Rockwell remarked that the effect of electricity was manifested when recomposition of the current took place. The body conducted electricity by virtue of its saline solutions. The skin was a bad conductor of electricity, and only when it was well moistened would it readily conduct. Therefore, placing one pole on the posterior portion of the body and the other in the anterior, the electricity, after it penetrated the skin, naturally, all other things being equal, took the nearest course from pole to pole. The diffusion of electricity over a brass ball had been advanced as a parallel case, but in that instance, as was well known, the electricity diffused itself over the surface and did not penetrate into the interior. The cases were far from being parallel, not only upon the theory of the conductibility of the body, to which allusion had been made, but because actual physiological experiment showed that the electric current did penetrate to the interior of the body. By experiment with the rheoscopic frog, Erb had found that the galvanic current could be made to pass directly through the brain. Neither the interior of the body nor the muscular substance external could be affected to any great extent by holding one pole in the hand and placing the other under the foot. What he objected to in Dr. Gray's method was that it did not answer the purpose of general faradization, because an incomplete could not take the place of a complete method of application. Certainly fifteen or twenty cases would not substantiate its value as a substitute for a method which had gone through years of experimentation both in this country and in Europe.

Dr. Hammond asked either Dr. Beard or Dr. Rockwell, if by any process of general faradization we could act upon the viscera of the body by means of the faradic current? Could the faradic current be made to act upon the heart? Dr. Hammond thought it was not possible by the use of the strongest possible faradic current in general faradization to so affect the heart that its influence on the pulse could be recognized by either the finger or by the sphygmograph.

Dr. Rockwell thought it possible to affect the heart, and stated that Fothergill claimed that electricity acted upon the heart, as proven by an increase of the strength of the pulse.

Dr. Hammond.—That is indirectly through its general tonic effects.

Dr. Rockwell remarked that he was positive he had seen it affect the heart, time and time again, independent of the general tonic effect produced through it upon the great sympathetic and pneumogastric.

On motion, the discussion was postponed until the evening session.

PATHOLOGICAL LESIONS IN THE NERVOUS SYSTEM IN YELLOW FEVER.

Dr. H. D. Schmidt, of New Orleans, demonstrated microscopical specimens prepared from the brain and spinal cord of patients dying of yellow fever.*

CHOREA IN HYSTERICAL CHILDREN.

Dr. L. C. Gray, of Brooklyn, read a paper containing the histories of several cases in which chorea and hysteria coexisted in the same children: three girls and one boy.

DISCUSSION.

Dr. Hammond referred to a case in which the movements were similar to those seen in the boy presented by Dr. Gray, and which yielded completely to large doses of arsenic. The remedy was gradually increased until the patient took $\frac{12}{40}$ of a grain of arsenious acid daily. The patient remained in the city two months, and then returned to her home in Texas. This spring the choreic movements returned; she came back to New York: was being again treated in the same manner as before, and the twitchings were measurably diminishing. In addition to the arsenic, she was taking valerianate of zinc.

Since he had used large doses of arsenic, he had not seen a case of chorea that he would be willing to abandon, until he had produced the toxic effects of the drug. There was no external sign by which he could tell whether the patient could bear the remedy or not, but had not had any difficulty in using it.

He was now ready to take back the statement he had formerly made that chronic chorea was not much amenable to treatment. Chorea in adults, however, was almost an incurable disease.

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* See, for a full account of Dr. Schmidt's researches upon the pathology of yellow fever, the *New York Medical Journal*, Feb., 1879, p. 113.

In the use of arsenious acid, he began with $\frac{7}{40}$ of a grain, three times a day; or *four* drops of Fowler's solution to a child, three times a day, and then produced the toxic effects of the remedy as soon as possible.

Dr. Gray referred to cases in which he had commenced the arsenic treatment by the use of ten-drop doses, and he had found that, as a rule, those doses at once were well borne.

Dr. Rockwell suggested the use of electricity in such cases —general faradization — because the children were usually delicate, alternating it with the galvanic current applied to the parts affected. Some years ago Dr. Miller, of this city, sent him two cases—mother and child, suffering from choreic disturbances of the face. The mother's had existed her lifetime, and received no benefit from treatment. The child's symptoms had existed more than a year, but were entirely dissipated by treatment, which consisted in the use of electricity and the subcutaneous injection of eserin, in $\frac{1}{28}$ grain doses. During last winter, he was called to see a younger brother, five years old, who was suffering in the same way. The first symptoms had been noticed several months previously. The same treatment was adopted, and was followed by prompt relief.

Dr. R. W. Amidon, of New York, then read an elaborate paper, entitled, "A Contribution to the Study of Cerebral Localization."

On motion, the discussion was postponed until the evening session.

The Association then adjourned to meet at 8:30 p. m.

THIRD DAY—EVENING SESSION.

The Society was called to order by the President.

Present: Amidon, Beard, Edes, Gray, Hammond, Miles, Putnam, Rockwell, Seguin, and Spitzka.

The President announced the committee on metallo-therapy: Drs. A. D. Rockwell and Wm. A. Hammond, of New York, Dr. J. C. Shaw, of Brooklyn, Dr. S. G. Webber, of Boston, Dr. J. S. Jewell, of Chicago, and Dr. Geo. Van Bibber, of Baltimore.

The discussion upon Dr. Amidon's paper on the "Localization of Cerebral Diseases," was continued.

Dr. E. C. Spitzka, of New York, remarked that the tendency had been for some time to localize lesions too closely, and he was pleased to notice that Dr. Amidon had appreciated the error of this method, and had recognized the anatomical variability of the brain.

Dr. Amidon remarked that in fœtal life the brain was first a mere vesicle, with perfectly smooth external surface, with nerves running to it. It was probable that in this smooth surface motor centres were already established. When the fissures began to appear and the sulci to form by foldings in of this outer membrane, the centres might be carried by such foldings sometimes in one direction and sometimes in another. In this way the motor centres, for instance, might be brought either in front or behind, or even at the bottom of the fissure of Rolando. A motor centre, therefore, could not be invariably referred to a particular convolution, but must be indicated by a pretty large area of tissue.

Dr. Seguin remarked that for some time he had recognized this fact, and had been in the habit of indicating the various centres by rather large areas, which occasionally overlapped each other.

CEREBRAL HEMORRHAGE.

The President (Dr. Miles) then showed the brain of a person whose history was as follows: He had been suffering from Bright's disease, and was suddenly taken with right hemiplegia of arm and leg. When first seen by the speaker, immediately after the attack, his mind and speech were perfectly normal. He soon began to recover the use of his arm; and later, in three or four weeks, movement began to return to the leg. Before recovery was complete, however, the patient died. Post-mortem examination showed the existence of a small circumscribed clot, partially absorbed, in the left hemisphere at a point where a section passing through the anterior frontal convolution downward, not quite parallel with the fissure of Rolando, would cut into it. It was a region supposed to be motor. The case was particularly interesting, however, because the clot was very circumscribed; it did not involve the cortex, but only the nerves running to the cortical motor centres, and there was no mental disturbance with it.

Dr. E. C. Seguin, of New York, related two cases showing abnormal sexual emotions, interesting chiefly in their psychological bearings. A female patient, perfectly well except for attacks of migraine, experienced occasionally the sexual orgasm while sleeping and dreaming; the dreams that excited the orgasm not being at all of an erotic character. On the contrary, the orgasm was generally excited by some emotion of great disappointment. At the climax of such disappointment the orgasm would be experienced. The other patient experienced the orgasm when she was greatly hurried in her dream. The cases showed an apparent violation of the law of association of ideas, and a variation from the ordinary way in which the orgasm was brought about.

Dr. Seguin also exhibited sections of a spinal cord in a case of myelitis, these sections showing the vacuoles, or dropsy cells, which had been referred to in previous discussions. The sections were made from just below the seat of greatest inflammation.

Dr. Spitzka related the case of a patient who had died from a cystic tumor at the base of the brain. The tumor was oval in shape and about the size of a small orange. Its exact nature could not be determined, but it probably originated from the dura mater. During its growth the patient gradually became blind, paralytic, and insane. Four distinguished neurologists had during life pronounced upon it four different diagnoses, not one of which was correct.

Dr. Spitzka read also the abstract of a more elaborate article upon

THE PEDUNCULAR TRACTS OF THE ANTHROPOID APES.

The object of the paper was to show the homologies that existed between these structures in man and the animals just below him. The field had not previously been worked up, and yet its study revealed greater evidence of structural affinities than the study of the hemispheres. The conclusions reached were, that the difference in those tracts between man and the anthropoids was less than that which often existed between the tracts in different men, and that there was in fact a structural identity between man and the ape next below him. The discussion of the paper was postponed till the next meeting of the Association.

Dr. J. C. Shaw, of Brooklyn, read a paper upon

TENDON-REFLEX IN THE INSANE.

He had examined 130 insane paretics in reference to this symptom. Ten of these had general paralysis, in whom tendon-reflex in 3 was absent. All the cases had a certain ataxia of gait. In the early stages of the general paralysis he had never found the reflex abolished. In 15 other cases of dementia, etc., reflex was absent. Two or three of these were cases of senile insanity. In 29 of the remaining cases reflex was slight: in 76 it was normal.

The conclusion arrived at was, that if tendon-reflex was present in paretics, degeneration of the posterior columns might be excluded.

All were agreed that the tendon-reflex was a reflex phenomenon; and we should expect a lesion in the lower part of the cord of a patient in whom tendon-reflex was absent, showing such a lesion. Other specimens were shown, illustrating the idea that in chronic meningeo-myelitis the changes were apt to be more marked and more frequently seated in the posterior portion of the cord, in this way abolishing tendon-reflex.

Dr. Spitzka stated that he had at one time made an examination of 40 cases; and his examinations, so far as they went, tended, like those of Dr. Shaw, to show that the abolition of tendon-reflex was an indication of a lesion in the posterior columns of the cord.

The Association then adjourned, to meet on the third Wednesday in June, 1880, in the city of New York.

Reviews and Bibliographical Notices.

I.—DISEASES OF THE SPINAL CORD.

Maladies du Systeme Nerveux. Maladies de la Moelle. Lecons Professees a la Faculte de Medicine. Par A. Vulpian. Recueilles et Publiees par Le Dr. Bourceret. Paris, 1879. Pages 512. (*Diseases of the Spinal Cord, etc.*).

The sixteen lectures of which this volume consists, constitute one of the annual courses delivered by Professor Vulpian from his chair of *Experimental Pathology*, in the Faculty of Medicine, in Paris. It is but a short time since we gave a lengthy notice of a work in two volumes, by the author of these lectures (*Lecons sur l' appareil vaso-moteur*). It was with no ordinary expectations that this last volume was taken up, and it is pleasant to be able to record the fact, that our expectations have not been disappointed. This latest of his works fully sustains its author's already brilliant reputation.

No other part of the central nervous system is so well understood as is the spinal cord. And no person now living, it is probable, has a more full and easy command of our present knowledge, whether relating to its healthy or morbid states, than has Professor Vulpian. But few scientific men know, as well as he does, the conditions of real proof, or any better understand the nature and modes of critical application of the criteria of a fact, in experimental physiology. Perhaps, also, none excel him, in capacity and experience, in the application of physiological data to the elucidation of pathological problems, within the difficult domain of the nervous system. In this, as in his preceding works, our author has brought face to face the results of experiment and of clinical observation with great candor and critical insight, and with highly satisfactory results. It is to a brief survey of these latter, as well as the methods for attaining them, that we would invite the attention of the reader.

In a former course, Professor Vulpian had discussed the degeneration and regeneration of nerves, the consecutive alteration of muscles, etc., secondary alterations of the spinal cord in consequence of the lesion of nerve trunks, reflex paralyses and reflex atrophies, reflex epilepsies, traumatic lesions of the cord, cicatrization of the cord with a restoration of functions, etc.

In the course contained in the present volume, we have first, compression of the cord considered, whether sudden or slow, whether produced by traumatisms or tumors of various sources and kinds, and of the differences of one kind as compared with others. Then next in order, primary lesions of the cord and its membranes, such as spinal meningitis, in its varieties; hemorrhages intra, or extra-meningeal; "necrobiotic softenings" of the cord, diffuse acute myelitis, in its different forms; interstitial myelitis, suppurative and hyperplastic; of systematic myelitis, acute and chronic, comprehending: locomotor ataxia, symmetrical sclerosis of the lateral columns, infantile muscular atrophy, progressive spinal paralysis, chronic myelitis *en ilots ou en plaques*, sclerosis *en plaques*, transverse sclerosis, etc., also tumors of the cord proper, and medullary hemorrhages are considered. Next in order a study is made of those spinal lesions which result from encephalic disease by descending degenerative disease, etc. To such important subjects is the course of lectures now under consideration consecrated.

But without farther preliminary, we pass to a notice of special topics. Then first of all "Compressions of the Spinal Cord." These are divided into *sudden* and *slow* compressions. The former are treated first in order, and are of course chiefly traumatic, or more widely speaking, accidental. There is as a rule disturbance of function in those parts of the cord which lie behind the point of compression. One of the most notable things is, the more or less complete temporary loss of reflex excitability behind the point at which the compression is exerted. In such cases reflex excitability is usually regained after a variable period, and, as might be expected, often rises above the normal degree of intensity.

At this point M. Vulpian makes a digression, to enable him to repeat and confirm the observation of M. Fubini, which was to the effect, that in sudden compression of the spinal cord,—say in the dorsal region,—he had seen the posterior lymph-hearts become very much slowed in their action, and he found it impossible to quicken their action by excitations of the sciatics or of the cord below or behind the compressed point. M. Fubini was led to conclude, that the spinal centre for the innervation of the lymphatic hearts in question has its seat high up in the cord, perhaps in the medulla. While M. Vulpian is able to confirm the facts he is unprepared either to accept or reject the conclusion of M. Fubini. The symptoms vary of course with the seat and degree of the compression.

M. Vulpian next passes to *slow* compressions of the cord. He repeats, from the writings of Charcot, the various kinds of disorder or disease which may produce it. The list is instructive, and is as follows:

1. By affections of the vertebræ. Under this head, first in rank is Pott's disease, and its various accidents, tuberculous caries of the vertebræ, spondylitis (inter-vertebral), cancer of the vertebræ, exostoses, vertebral gummata, and other neoplasms of the vertebral column.

2. By extra-vertebral tumors, such as aneurisms, cancers, hydatid cysts, prevertebral abscess, (retropharyngeal abscess,) opening either into the vertebral canal, or by finding its way slowly through the openings in the vertebral column.

3. By lesions of the vertebral ligaments, as in abscess, tumors, cysts, etc., developed in the tissues, immediately investing the dura.

4. By affections of the dura, causing exudations; tumors, such as sarcoma, myxoma, osteoma, cancer, psammona, and abscess, hydatids, etc.

5. By disease of the pia mater, causing exudations, and various morbid productions arising from this membrane.

6. By tumors of the cord itself; gummata, tubercles, cancer, sarcoma, glioma, etc.

7. By disease of the central canal of the cord, of which encysted hydrorachis, and slow intra-medullary hemorrhages are samples.

M. Vulpian has chosen the slow compression of Pott's disease as the type of its kind. How is the compression produced in this disease? The until recently classical notion, that it is due merely to deformity of the vertebral column, is rejected for the view arrived at by Echeverria, and by Michaud. It may be due to abscess at the seat of bony disease, or to caseous exudations at the same point, or as MM. Echeverria and Michaud suppose, to exudations from the dura at the point where the vertebral disease is, when though a paralysis is present, yet there is no deformity, such as kyphosis. The compression then is produced by the exudation in the site of a true *pachymeningitis extra.* The meningitis is the consequence of an extension of disease, from the diseased bone outside, to the dura inside, and from this arises the exudation, which in its turn causes the compression. It is a sort of papilloma, or a caseiform growth, according to M. Michaud, who has made such cases an independent and fruitful study. Such is the more common cause of compression in Pott's disease.

In the motor paralysis which occurs in such cases, one of the peculiarities consists in the rather complete preservation of sensibility, especially that of touch. In paralysis from Pott's disease, the æsthesodic tract of the cord is respected in a singular way. In seeking to explain this state of things, it would be natural to assume that it depends on the fact that the disease of the vertebræ is in their bodies and the joints between them, and hence the swellings and growths which extend from the seat of disease so as to press on the cord, would naturally first reach its front aspect, or in other words the motor tract. But it is not equally true for the posterior columns, which are not pressed on, and hence the absence of sensory paralysis. This reasonable explanation is rejected by M. Vulpian. His idea seems to be, that if a tumor advances against the cord from the bodies of the vertebræ, and hence presses against its anterior columns, that the pressure will carry the cord back against the posterior wall of the spinal canal, so as to compress it at last, the same behind as in front, and hence the loss of function in the posterior part of the cord should be the same as that in the anterior columns, which, however, is not the case. But it seems to us M. Vulpian has neglected to consider certain conditions of the case, which should not be overlooked.

In the first place it should be remembered, that motor paraplegias of the kind now being considered, are seldom complete. Then it is our opinion that M. Vulpian has over-estimated the degree of the backward displacement and of the compression of the cord, in the slow compression paraplegias of Pott's disease. The displacement backwards is less, and compression needful to produce this paraparesis far lighter than may be supposed. But the chief fact which seems to have been overlooked, is the resistance by the *ligamentum denticulatum* on either side to displacement of the cord. It would really require considerable compression of the cord, from before backwards, to so far overcome its anchorage by the *ligamentum denticulatum* as to permit of its compression behind on the firm membranous and bony posterior wall of the spinal canal, and hence in the same way produce a sensory paralysis of the same degree as the motor. A degree of pressure on the front of the cord quite sufficient to produce a motor paraplegia might be easily produced, and yet leave the posterior columns free in their functions, because free from solid pressure. Then again, the fact that tactile sensibility is most frequently preserved, can be readily understood if the above views are accepted, and if Schiff's opinion is correct, that the channels for tactile impressions in the cord are in the posterior columns. At any rate we feel that M. Vulpian's explanation of the phenomena is lame and inadequate, and his rejection of the simple explanation we have referred to was made without due consideration. We have elaborated this subject at some length, because it is important and is easily misconstrued.

M. Vulpian raises the question as to the sensibility of the dura mater spinalis, and clearly adopts the opinion, on experimental and clinical grounds, that it is sensitive, and under certain circumstances of disease, exquisitely so. We call the attention of the reader to this subject, not because it is novel, but rather because of its practical importance in spinal disease, especially in the presence of spinal pain. In our opinion, the dura is much more often affected than seems to be suspected by the majority of physicians.

M. Vulpian considers the preservation of reflex excitability of the cord, below the compressed point, for long periods, to be an important diagnostic point, though consistent with other forms of localized disease of the cord proper.

In the fourth lecture M. Vulpian considers certain circulatory and nutritive affections resulting from Pott's disease. Passing by the vaso-motor disorder in parts innervated from or below the seat of disease of the cord, we come to note certain other phenomena, and among them the trophic disorders sometimes witnessed in such cases. Besides the sudden appearance of bed sores, there are various neuroses of the skin observed, such as zona. But among the few instructive instances on record, those of Baerensprung, (*Charite Annalen*, X., XI., XII.) and Sattler (*Stricker's Med. Jahrbuecher*) not to mention others, are omitted.

In noticing the arthritic affections which arise in some cases from the spinal disease, the credit of first recognizing the relation of the joint affections to the spinal, is given to Dr. S. Weir Mitchell, when as we understand the matter, it should be given to his father—Dr. J. K. Mitchell.

In discussing *cervical paraplegias*, M. Vulpian shows that it can be produced experimentally, by slight mechanical compression of the anterior face of the cervical cord.

But how it is produced clinically is a more serious question. He passes in review the various hypotheses devised to explain the occurrence of this curious form of paraplegia, so far as known to him, and rejects them, to adopt, substantially, the view that the reason why the arms, and not the legs, are paralyzed by the lesion of the cervical cord, in such cases, is because the paralysis is due to compression of the grey rather than the white matter of the cord. No special stress is laid on this explanation by its author. But this view does not agree with the results of the numerous experiments and clinical observations on this subject, of Rosenthal (*Stricker's Jahrbuecher*, 1876, 381–400, JOURNAL N. & M. DISEASE, 1877, p. 82. etc.), whose observations are not mentioned by Prof. Vulpian. In speaking of some ascending and descending degenerative diseases of the cord, and the possibility of experimentally producing them, the interesting fact is developed, that certain secondary spinal lesions which can be produced in the dog, cannot be produced by the same means in the pigeon and the guinea pig. Such facts have singular significance, in view of the discordant results arrived at by various experimenters, who have operated on different animals by means of the same agents. In this connexion we notice what must be a misprint, that is the statement that the degenerative atrophies of the lateral columns of the cord, are *ascending*, when the contrary is true. Prof. Vulpian does not consider "fulgurant" pains, and "contractures" of the muscles of the members, an evidence of descending degenerative disease of the lateral columns, so much, but rather as dependent on localized myelitis.

M. Vulpian calls attention to the paralysis produced by cancer of the vertebral column. He declares that often it makes itself known by no symptoms, save those which arise from the resulting compression of the cord. There is often no pain, or other local symptoms to denote the presence of the cancerous growth. But in others in which the cancerous affection is more extended, there is said to be exquisite pain, in so far that it is said such cases are types of painful paraplegias. Tumors of the tissues investing the dura, and of the dura itself, such as sarcomas, myxomas, psammonas, tumors of the vertebræ, (exostoses, gummata,) tumors of the cord, gliomas, gummata, tubercles, sarcomas, cancer, etc., produce similar phenomena it is said, and when they produce compression of the cord, they give rise at the point compressed to interstitial myelitis.

Compression of the *posterior face* of the cord produced more often motor than sensory paraplegia.

In discussing the results of unilateral compression of the cord, and among the rest the alleged loss of sensibility in the side of the body opposite to that compressed, M. Vulpian refers to his paper in the *Dict. Encyclopedique des Sciences Med.*, on the spinal cord, for a partial refutation of the notion (Brown-Sequard,) that sensory conductors decussate soon after entering the cord. But though we have read this part of M. Vulpian's writings attentively, we cannot share at present, to any considerable extent, his opinions. It still seems to us, that the bulk of evidence supports the view which affirms that the conductors of sense impressions decussate soon after entering the cord, at least, in great measure. The great mass of motor fibres do cross over, in the medulla or near it, from one side to the other. But certain bundles do not cross over at this point. They pass down in the same side of the cord as that of the brain in which they originated; it seems probable, chiefly in the *direct pyramidal tract* of the anterior column (column of Tuerck) and to cross from one side to the other, in the anterior white commissure, to terminate in the anterior horn of grey matter of the opposite side. These fibres of the direct (motor) pyramidal tract cross over more freely, perhaps, in the lower three-fourths of the cord than in the upper fourth. By this means we may explain how it comes to pass, that in brachiocrural hemiplegia from unilateral lesion of the cervical cord, that the arm is more completely paralyzed than the leg. This subject is discussed at some length by Prof. Vulpian; and his view of the spinal mechanism through which the above-mentioned paralytic phenomena come to pass is substantially as we have given it.

The remainder of this quite lengthy lecture is devoted to a recital of cases, found chiefly in the service (Salpêtrière) of M. Vulpian. But though of much interest to the neurological student, they cannot be profitably referred to in this notice.

The next lecture comprises but eight pages, while the fourteenth lecture, devoted to locomotor ataxia, is *one hundred and seventy-seven pages* in length, and no inconsiderable portion is in small type! It would require *not less than four* consecutive hours for its delivery. It is difficult to believe that M. Vulpian ever gave a lecture of such length, or that any class of students would sit and hear, or if they did hear, could comprehend and remember it.

We mention this extraordinary disparity in length between certain of these lectures to give ourselves the opportunity to notice the common practice of publishing lectures in which the reader is left to suppose that they were wholly extemporaneous, and were reported phonographically, the busy author not finding time to do more than make a few hurried corrections of the notes of the reporter; and yet the lecture often bears all the marks of careful elaboration, or, as in the present case, possesses characteristics utterly at variance with, to say the least, an average experience in such matters.

If one must publish a lecture which was never delivered, or not as it was delivered, either let the proprieties of the case be observed, or a frank explanation be made of its anomalous features.

We would not wish to be understood as specially censuring the course of M. Vulpian in the matter under discussion, but we have felt we ought not to let the occasion pass for noticing some of the unpleasantly suggestive characteristics of too many of our published lectures, whether in periodicals or books.

But we cannot, in this notice, follow M. Vulpian's admirable and instructive book any farther. To adequately examine it, and extract passages of interest to such of our readers as may not meet with the work itself, would be to us a pleasant but a long task. We have intended from the first to examine with some care only the first few lectures, that we might show our readers something of the valuable data and of the suggestive and critical reflections on the same with which the work abounds. More than this we cannot do at present.

It is the avowed intention of the author to continue his studies until he has produced a complete work on the nervous system, in its clinical aspects, in which studies all the resources of anatomy, whether healthy or morbid, and of physiology, whether human or comparative, are to be brought to bear in the hand of a master on the clinical and pathological phenomena of nervous disease, which must continue for a long time in the future, as in the past, to challenge and perplex even critical and cautious observers such as is M. Vulpian. We are afraid the time is yet distant when works of this character will be much in demand by the profession of this country.

II.—ANATOMY OF THE NERVOUS CENTRES.

Anatomie des Centres Nerveux. Par le Professeur G. Huguenin. Traduit par le Dr. Th. Keller; annoté par le Dr. Matthias Duval.

The appearance of this translation is an evidence of the widespread interest which is being manifested by the French for the results obtained by German cerebral anatomists; and since the original owed its chief importance to the fact that it popularized, so to speak, the views of Meynert, the translation may be considered a tribute to the ingenious deductions of the latter author, the recognition of whose services to the cause of *applied* cerebral anatomy had its way paved by no less a one than Charcot.

Inasmuch as the translation has had added to it numerous annotations by a French investigator, its criticism naturally falls into two parts—the first relating to the original, the second to the corrections, explanations and comments of Dr. Duval. As to the translation as a translation, we can only speak in high commendation; the French rendition is both correct and above all fluent, and as free as every good translation should be.

Among the pupils of Meynert, Huguenin has become to be considered a leading representative, from the mere fact that he was the first to give to the medical public a simplified version of his teacher's "projection theory." In fact, Huguenin's book may, aside from some abstractions from Koelliker's embryology, and Gratiolet and Leuret, be considered a rather faithful transcription of the lectures delivered by Meynert at the Vienna University. This resemblance extends so far that we even recognize in the diagramatic representations of Huguenin's work most of the blackboard drawings which that master employed to illustrate those lectures. From the fact that the original contained no clear references to the sources from which the more elaborate illustrations were obtained, it is to be excused that the French translators have equally failed to indicate them. They have, as it is, improved on the original in so far as they furnish the description of the figures at the bottom of the respective pages, to the great convenience of the student.

Since many of the views presented by Huguenin have since, or even before the appearance of his book, been shown to be faulty, it may be well to call attention to some of the more prominent errors:

Page 1. The author states that the parts on each side of the primitive groove are the medullary laminæ, speaking of the origin of the nervous central axis as originally double, and confounding the primitive groove and medullary gutter. The medullary lamina is single and axial *ab initio*, and the groove and tube formed by its incurvation have nothing to do with the primitive groove!

His views on the disappearance of the embryonic roof of the thalamencephalon are likewise antiquated and incorrect. And it has now been shown that the spinal ganglia are not derived from the mesoblast (p. 15), but from the medullary tube itself. (Balfour, Shenk, Götte and others.)

As far as the rendition of the projection system is concerned, Huguenin has naturally adopted all the errors of Meynert's first publications, without a word of that correction which justice to the researches of Gudden and the most eminent of Meynert's own pupils, Forel, would seem to demand. Thus the erroneous and utterly unfounded view, that the oculo-motor nuclei are under the direct innervation of the cortex, is reproduced. The thalamus is stated to be a reflex centre, a view which was never expressed by Meynert in this crude way: the latter considered it a presiding ganglion of the higher automatism.

On page 26 it is stated, evidently in the haste of transcription, that the corpus restiforme goes into the *anterior* columns of the cord ! ! ! That this gross clerical error escaped the annotator is remarkable. At the same place the enumeration of the cerebellar peduncles is so confused that one would from the reading be led to infer that the *brachium pontis* and the transverse fibres of the pons were two different systems, whereas they are but one system.

In describing the gyri and sulci of the hemispheres, the system of Gratiolet is the one chiefly followed, and the more modern researches are but briefly dwelt on.

Figure 57, illustrating the anatomy of the fourth ventricle, is doubly incorrect. It makes it appear as if there were a special nucleus between the median fissure, striæ medullares and *ala alba medialis*, while the anterior half of the floor is still more inexact, and unlike anything ever found in nature. There is a great lack of clearness on Huguenin's part, where he speaks of the olfactory bulb and lobe without exhibiting their different location in any of his figures. The view that both the anterior and posterior pair of the corpora quadrigemina are centres of vision, as maintained by Huguenin, has now been totally exploded: Gudden having shown that extirpation of the eye in the new-born leads to atrophy of the anterior pair alone; Forel having demonstrated that no optic fibres enter the posterior pair, which is well developed in the blind mole; and we having called attention to the fact that in reptiles, in whom, contrary to current views, the posterior pair is present, these are developed independently of the anterior pair, and in demonstrable relation with none of the optic fasciculi.

The tract of the lemnisci, as given by Huguenin, is also highly problematical. Through carelessness one of the wood-cuts given to illustrate their course, is given as inverted, and Fig. 103, lower down in the series, makes the vertical (pyramidal) fibres of the pons appear as if they coalesced, thus giving, unintentionally of course, a wrong impression. The term, "*superior* root of the trigeminus," is certainly inapplicable to the bundle so designated in figure 110.

Unfortunately, Prof. Huguenin, while he has not failed to introduce a large number of figures to illustrate the superficial anatomy of the brain in lower animals, has neglected to employ such as are calculated to elucidate the predominance of the *pes pedunculi* in man as contrasted with the tegmentum. A foot-note of Dr. Duval makes up for this defect, but of course only partially.

The very plausible diagram on page 203 loses its value in view of the incorrect assumption of an origin of the optic tracts in the posterior pair of the corpora quadrigemina, referred to above. In Fig. 119 the extent of the trapezium in the baboon is exaggerated, and the olivary eminence diminished, while in Fig. 121 the sheep's trapezium is represented as smaller than it is in reality. Some of these imperfections are of course of no great importance, and in so far as the book was intended to serve as a guide to the beginner, it might be expected that the annotations of Dr. Duval would correct all possible misapprehensions of the actual state of cerebral anatomy by giving the most recent results in his foot-notes.

In their introductory remarks the translators state that they have not quoted the writings of Flechsig and Forel, because these authors had offered few or no positive results. We think that negative results are under occasion as important as positive ones; and we consider it as detracting seriously from the value of this translation that the very decisive researches of Forel on the fallacy of the oculo-motor and visual innervations, as given by Meynert, were not presented *at least* as current views entitled to *some* consideration!

On page 2, the first annotation, although written in misapprehension of the text, serves to correct its errors, and proper views as to the relations of the medullary groove referred to are given. The next annotation states that the cornu ammonis and the "trigone" are one and the same thing, merely on the ground that the fibres of the fornix are derived from the cornu ammonis! On the same grounds the author might say that the cerebral cortex and the internal capsule were "qu'une seule et même chose." The charming naiveté of the French appears in the statement that the study of the gyri of the hemispheres is the "order of the day."

Duval's idea that the crucial sulcus of the dog is the analogue of the perpendicular occipital of man, would be untenable for a moment, even if Meynert had not shown the true homologies from the bear's brain. He neglects to point out, while insisting that the nucleus oculo-motorius and n. trochlearis are distinct, that their molecular grey matter is continuous. If the annotator's ingenious views regarding the origin of the intermediate nerve of Wrisberg, presented on page 211, is correct, then the physiological and anatomical problem of the gustatory innervations is solved.

To the error regarding the olivary nucleus dentatus, an article in the JOURNAL OF NERVOUS AND MENTAL DISEASE, has already called attention. We would not refer to it here were it not that the annotator has during the last few years repeatedly proclaimed his alleged discovery, that the animals inferior to the quadrumana in the scale had no *nucleus dentatus*. They have unquestionably, and Duval's statement is based either on imperfect or fallacious observation.

On page 247 a very useful annotation is made to clear up the metamorphosis of the cord into the medulla oblongata. It is unfortunate that the author has inverted all the eight figures so as to make the ventricular floor the inferior aspect, and the basilar parts the superior. This he has done to accommodate the cerebral sections to the spinal ones figured in the conventional way. Now, as it was easier for Mahomet to go to the mountain than for the latter to come to the former, so it would have been far simpler to invert the spinal sections from their customary and incorrectly figured position, than to turn the entire brain upside down.

On the whole the work will prove more useful to him who desires a general view of the cerebral architecture than to him who desires correct topographical information. In presenting it to the French medical public the translators have done a service which cannot be undervalued. The views of the cerebral functions, their complexity, and the manifold tracts subservient to them, which have been promulgated by Meynert, will serve by becoming familiar to the French medical public to act as a healthy antidote against the vagaries which, whether in the shape of the "nihilism" of Brown-Sequard or the metallo-therapy of his great opponent, can only be founded on an imperfect conception of the nervous mechanism.

While we do not hesitate to insist that in many respects the work of Huguenin is faulty, and that its translators have failed to correct some of the more serious errors, it must be admitted that there is no better *readable* version of modern cerebral anatomy in the market than the present one. We may hence be permitted to hope that a still better and safer guide to the cerebro-spinal anatomy will be furnished to the American reader in the forthcoming American edition, which by more careful notes, better illustrations, and above all, a due reference to cotemporary researches, will avoid the errors as well of the original as of the translation before us.

E. C. SPITZKA.

III.—HANDWRITING: ITS PHYSIOLOGY AND PATHOLOGY.

Die Schrift: Grundzuege ihrer Physiologie und Pathologie. (*Handwriting: its Physiology and Pathology.*) Von Dr. A. Erlenmeyer. Stuttgart: Adolf Bonz & Co., 1879.

In his preface the author informs us that he has collected specimens of handwriting of patients for some years, and that on reviewing them for publication, so many questions turned up that he was forced to prepare some physiological ground for the subject; hence its publication in the form of a brochure of some 70 pages. The book commences with some reflections on the cause of the direction of script. The habitual writing of European nations from left to right, the author attributes to the greater ease with which motions of abduction can be performed as compared with adducting movements, since in abduction the trunk is not in the way. But this view, it seems to us, is not borne out by every-day experience; not even in the instances mentioned by Erlenmeyer, such as piano-playing and turning of a crank, movements which, according to him, are more easily executed in abduction than in adduction. But Erlenmeyer argues further that left-handed people ought hence to be able to write more easily from right to left, and cites in proof the ancient Jews, whose script had this direction. A questionable passage in the Talmud is interpreted by him to imply the frequency of writing with the left hand in those days. In a spirit of speculation difficult to understand, he deduces from the above assertions conversely that our right-handedness is acquired by writing a script running from left to right. The first chapter concludes with some remarks on "reversed writing" (Spiegelschrift). Buchwald has recently shown that patients suffering from right-sided hemiplegia write backhands (*i. e.* reversed) unconsciously when told to use the left hand, so that their script can be read best with the aid of a mirror. Erlenmeyer cites some cases, and adds that the same tendency exists also in all children when writing with the left hand. The speculative tendency embellishing this first chapter is fortunately not to be found in the rest of the book, which is written on the whole on a more substantial basis.

In the following chapter the author condemns the usual school directions ordering children to perform the writing movements with the entire hand, instead of moving the fingers alone, which latter motion, he claims on good ground, is much less fatiguing. Another point insisted upon is the fact that the most natural way is to write, not in a straight line, but in a curve, the radius of which is the forearm resting upon the olecranon as the centre. Writing in a straight line requires a constant correction of the natural tendency, which not all persons are able to achieve.

Script is composed of: 1, up-strokes; 2, down-strokes; and 3, curved lines. 1, up-strokes require movements of extension. The muscles involved are the extensor dig. comm., innervated by the radial nerve and the interossei (of the first two fingers) actuated by the ulnaris, while the thumb is kept in position by the united action of the extensor poll. long., opponens and adductor pollicis muscles (radial, median and ulnar nerves). 2. The down stroke is a movement of flexion (flexor digit comm. profund.). The thumb is at the same time pulled upon by the flexor poll. longus, while in sweeps the hand itself is flexed by the flexor carpi ulnaris. The nerves involved are the median and ulnar nerves. 3. The curved strokes require the alternating action of the above muscles, and besides, according to the direction of the curve, the abductors and adductors of the fingers. Since all nerves of the forearm are called into action during writing, the isolated paralysis of one of them will not render one set of movements impossible, but will be manifested in writing in all strokes. Paralysis of the radial nerve alone would not show itself in the down strokes.

Since the mechanical movements required in writing are the same in all persons, one might expect no difference in different handwritings. In fact there is very little when children first begin to learn writing. It is only in later years that the mental traits of persons influence their handwriting. The changes in handwriting due to morbid states the author divides very properly into, (1) mechanical, and (2) psychic alterations. The former variety refers only to the execution of the letters; its subdivisions are the (*a*) ataxia, and (*b*) the tremulous handwriting. The psychic alterations of writing are on the other hand characterized by errors in spelling and grammar. They are classified by Erlenmeyer as (1) those dependent on a conscious impulse (bewusst zwangsartigen) (*a*) Agraphia, and (*b*) Paragraphia, and (2) the voluntary, but unconscious variety (willkurlich unbewussten)—the writing in general paresis. Writers' spasm is not included in this list, since it either appears as one of the mechanical disturbances or renders writing altogether impossible. The term *ataxia* is applied to handwriting by the author in the same way as we are accustomed to speak of an ataxic gait. The writing is characterized by awkward and ill-proportioned execution of the letters; it is hence more or less illegible. Normally this form of writing occurs when the child first begins to learn writing. It is in that case the result of imperfect co-ordination of the movements. A similar inco-ordination results from affections of the cerebrum or cerebellum, or spinal ataxia, located in the cervical cord. The reconvalescence from intense infectious fevers disturbs the writing in the same manner. Intense fatigue can produce the same result, as can be easily shown by writing after over-exertion. Acute intoxication with alcohol or chloral will also render the writing ataxic.

The *tremulous* handwriting is not necessarily illegible. It is characterized only by wavy lines instead of straight strokes. It occurs normally in old age, as well as in all conditions of tremor. All persons write with a trembling hand when subjected to cold. This form is also quite marked in cases of chronic alcoholism before the victim has obtained the desired draught. This writing is to be expected in disseminated sclerosis, rhythmic chorea and paralysis agitans. The tremulous movement may take place in any one plane. If they occur in a plane perpendicular to the paper, the writing must consist of dotted lines. This form can be readily produced in the normal person by tetanizing the radial nerve and extensor digit. communis muscles by an induction current while writing.

In the *psychic* disturbances of writing the execution of the letters may be perfect, but there are errors in spelling and grammar, or the sense may be wanting altogether. In *agraphy* and *paragraphy*, the patient knows that he is writing incorrectly, but cannot help it. In *general paresis*, on the other hand, the patient writes incorrectly also, though not in the same manner at different times, but claims that he is correct. The former disorders are considered in a very lucid manner, the same mode of reasoning being applied in the analysis of writing which Kussmaul uses in the analysis of speech. While we cannot follow the author into his details, some idea of his considerations can be obtained from the following classification:

1. Ataxic agraphy—the incapability of co-ordinating the letters distinct from the mechanical disturbance.
2. Amnesic agraphy—the loss of the mental images (*Erinnerungsbilder*) of the different letters.
3. Paragraphy—the impossibility of adapting the corresponding letters to the conceptions (Vorstellungen).

A schema of the brain paths concerned in writing is here introduced. But while following the author through this pleasantly written part, we are not to forget that this is also purely hypothetical. The writing in general paresis becomes complicated as a rule with one or both forms of mechanical disturbances in the latter part of the disease. It is crowded with errors, but these are not due to the impossibility of writing otherwise; the patient can readily change them, and if sufficiently attentive will even correct them. The writing in general paresis bears the stamp of the disease so characteristic in the different movements.

In the last chapter Erlenmeyer sums up his results in order to estimate their importance for practical purposes. His conclusions we can state verbally:

1. The writing permits an early diagnosis of an organic diffuse affection of the cortex.
2. It confirms the differential diagnosis between the above disorder and a merely sympathetic cortical affection.
3. By means of the writing we can determine whether every case of general paresis is of a syphilitic origin.
4. Writing enables the physician to prognosticate with certainty the cause of general paresis.
5. The writing is an objective test for the therapeutic success consequent to certain drugs or the application of the constant current; it reveals also the psychic improvement in spontaneous remissions of certain brain diseases.

Conclusion No. 5 can be accepted readily by every reader, but the other statements are by no means adequately supported by the text of the work or the cases given in illustration. Like so many writers of monographs, Erlenmeyer is apt to overrate the import of his subject. But with the exception of those parts already referred to as rather speculative, the monograph is on the whole written in a very lucid, cool manner. The classification adopted by the author may appear artificial on glancing hastily at the book, but a careful study of the excellent plates appended will convince any one that the grouping of the disturbances of writing is well founded. On the whole a clinicist of experience will hardly find any direct clinical statement in the book which is entirely new, but the arrangement of the subject, which, by the way, has never been handled similarly, as well as the sound discussion, render the book worthy of careful reading. H. G.

IV.—ACUTE ASCENDING PARALYSIS.

I. Recherches sur les Lesions du Systeme Nerveux dans la Paralysie Ascendante aigne. Par le Dr. J. Dejerine, Ancien Interne des Hopitaux le Paris, Member laureat de la Societe Anatomique (Prix Godard, 1879). Paris, 1879. (*Researches in regard to Lesions of the Nervous System in Acute Ascending Paralysis.*)

II. Contribution a l'Histoire des Nevrites—Nevrites Disseminee. Par le Dr. J. Gros, Ancien Interne des Hopitaux de Lyon. Paris, 1879. (*Contribution to the History of Neuritis.*)

Since Landry, in 1859, first directed attention to them, a class of cases of paralysis have, in great measure, been a puzzle to pathologists. At first it was hardly credited that they constituted a distinct disorder, and they were considered by most writers to be only peculiar instances of rapid myelitis, to which they have much resemblance in their clinical features. But carefully reported observations by such observers as Vulpian, Hayem, Westphal, and others, have demonstrated, at least approximately, that they deserve to rank as a distinct affection, pathologically and even clinically separate from any ordinary forms of myelitis or other spinal disorder. The two monographs, the titles of which head this notice, are among the latest contributions to the literature of the subject, and as such they deserve our attention.

M. Dejerine's memoir is a critical study of the pathology of the disorder. He first opens with a historical review of the subject, in the course of which he also lays down the plan on which he proposes to treat the subject. He shows how, in late years, while the researches of Charcot, Vulpian, and others, at the Salpêtrière had elucidated the pathology and history of myelitis, that a few careful observations by Pellegrino Levi, Petitfils, Hayem and Westphal, in which careful microscopic examinations were made of the cord, and no lesions discovered, have given ground for the recognition of Landry's disease as a distinct morbid species. Some observations he rejects as imperfect, such as those of Chalvet and Calastri, and all those in which the case terminated otherwise than fatally, and with an autopsy. He believes with Vulpian, that there is no proof that these latter were actually cases of acute ascending paralysis of Landry, assuming that no well authenticated case of the disease has terminated in recovery; we shall have occasion to notice this manner of view farther on.

M. Dejerine begins his second chapter with the statement that he had been led to give special attention to the spinal nerve roots and with a brief account of his methods of microscopic preparation. His memoir is essentially an anatomo-pathological one, and the clinical features of Landry's disease are not its main objects. Nevertheless, these features form the subject of this second chapter, which is made up chiefly of the history and discussion of two cases of the disorder; one a personal observation of the author, now for the first time published, and the other a re-statement of a case already reported by MM. Dejerine and Goetz, in the *Archives de Physiologie* for 1876. These cases leave little or nothing to ask for in regard to completeness of the clinical record; this is especially the case with the first, which the author considers a typical case of acute ascending paralysis. He sums up its clinical history as follows: "The patient, without any pathological antecedents, entered the hospital on account of a certain degree of weakness of the legs, of which he had been complaining for a few days; this motor trouble was so little apparent that we first suspected malingering. After a few days, however, there was observed a slight choreic condition in the execution of movements, then, suddenly, seven days after his admission, he was seized with paraplegia, at first slight, then becoming complete, it ascended, gained the trunk and upper members, and proved fatal in seven days from asphyxia. During the whole duration of the disease the sensibility remained absolutely normal; no trophic skin disorder, no paralysis of the sphincters, no fever, save on the last day. Very well-marked diminution of electric contractility."

We might as well here give our author's general description of the disorder which follows the discussion of his second case, and presents a clinical picture of the affection as M. Dejerine understands it. He says: "In these two cases we have to do with individuals in previous good health, who are seized suddenly and without any appreciable cause, with weakness of the limbs. This paraplegia presents the characters that struck Landry's attention, and which have been observed by nearly all of his successors. At first slight, it rapidly increases and becomes complete; it is a flaccid paralysis without contractions or any exaggeration or abolition of reflex movements, which are generally normal as in our cases. We cannot, by the brusque flexion of the foot, produce any of the phenomena studied in France under the name of 'reflex tremor of the foot.' * * * The percussion of the patellar or Achilles tendons causes none of the special reflexes studied in Germany in later years by Erb and Westphal. In a word, the paraplegia is absolute, without appreciable exaggeration of any reflex phenomena.

"Then after a longer or shorter time, generally within one or two days, the paralysis gradually ascends, it reaches the muscles of the abdomen, the thorax, the arms, the respiration becomes more and more exclusively diaphragmatic, the diaphragm itself becomes involved, and the patient dies asphyxiated in a few days from the beginning.

"This rapidity of the progress of the paralysis has been noticed by all observers; it is sometimes astonishingly rapid, and causes death in two or three days. As in other previous observations, we note in our two cases the absolute retention of the sensibility of all forms, tact, temperature, pain; this symptom is of great specific value, and in the majority of the cases published to date, we have always found this function unimpaired or very slightly affected. * * * The skin and the cellular tissue are intact during the whole progress of the disease, there is no œdema, none of the cutaneous trophic disorders so frequent and rapidly developing in acute myelitis.

"Finally, as regards various viscera, we have met with no symptom that could be attributed to a medullary affection. The temperature in one of our two cases was normal during the whole course of the disorder except on the day of his death; in the other it varied each evening, some tenths of a degree above the normal."

The post-mortem examination in each case revealed nothing abnormal in the cord or medulla, but the anterior spinal nerve roots were found more or less altered or atrophied, and in the first this could also be traced in the intra-muscular nerves; the posterior roots were healthy.

Having thus given an account both historical and clinical of the disorder, M. Dejerine devotes the remaining portion of his memoir to its pathology. The results of examinations, so striking for the absence of spinal, and the presence of nerve-root lesions, are taken up and discussed with all the light that can be thrown by the researches of others as found in medical literature. First is asked the question, Is the alteration of the nerve roots, which is found in these cases and which is identical with the degeneration of the Wallerian section of nerves, merely a passive process or a neuritis? He concludes that the latter is the case, and not an interstitial, but a parenchymatous neuritis, and adopts the view of Waller and Vulpian, that the trophic influence for these anterior nerve roots is derived from the anterior grey cornua of the cord, rather than that of Cl. Bernard, who held that the spinal influence was inhibitory and modulating instead of exciting. But the nature of the special lesion itself is not the most important matter; how it is brought about is the most important question of the pathology of the disease. And here comes in the question, that has been discussed for a number of years, especially in Germany, as to the existence of a genuine disseminated neuritis, a *neuritis migrans* passing from the nerve to the cord and from nerve to nerve, not preserving its continuity necessarily, as an actual inflammation at all points. M. Dejerine considers this an un-demonstrated hypothesis, at least so far as a generalized disseminated neuritis is concerned. An observation of Eichhorst's, which that author had called a case of *neuritis migrans* or neuritis ascendens, is quoted and discussed. It appears to us to be simply a well-marked case of Landry's disease, but with some peculiarities, such as sensory involvements that separate it from the usual type of cases of this disorder. The cord and brain were found apparently intact, but the nerves of the arms were found to be in a condition of active congestion; the anterior roots and the peripheral nerves of other portions of the body were not examined. The case is in itself incomplete, but it is suggestive, and there is nothing but purely negative evidence, and that due to its imperfect examination, to prevent its being classed as a case of acute ascending paralysis. Our author is therefore, we think, correct in his judgment as to the proper reference of this case.

The discussion of the facts and theories of neuritis and their application to pathology in the cases of atrophies, reflex paralyses, etc., is close and full, and the author concludes, himself, that the only plausible hypothesis to account for the degenerations in the nerve roots met with in these cases of ascending paralysis is that of an alteration of the anterior grey matter of the cord. But he leaves us in doubt as to what the alteration may be, favoring, however, the idea that it is a purely dynamic one, modifying the trophic action of the centres in the anterior grey horns, instead of, as he says, an ascending neuritis. The caution with which he guards his statement of belief in the alterations of these anterior horns, seems to us needless. The possibility of a protopathic neuritis affecting the anterior roots simultaneously without participation of the cord, is too great a tension on a reasonable imagination to be admitted, according to our view of the case.

The theory of a general toxæmic condition developing itself in Landry's disease, especially on the cord, that has with certain reservations been supported by some authors of note, such as Hayem and Westphal, is not credited by M. Dejerine, who could discover no evidence of any toxæmic conditions in the two cases that came under his own observation. The clinical features of the disease as well as the revelations of the autopsy exclude myelitis; the affinity with anterior polio-myelitis that has been maintained by some, is likewise discredited on account of the absence of anatomically demonstrable alterations of the anterior spinal horns. The author adds, that there is in the clinical progress of ascending paralysis a capital point of difference between it and myelitis of the anterior horns: that is, its constant fatal termination. We would only say in regard to this, that if, as M. Dejerine seems to hold, an autopsy is essential to confirm the diagnosis, it is too much to say that the clinical history affords adequate means to its recognition. As stated earlier, he rejects all reported cases that terminated in recovery from the category of Landry's disease; a practice common enough among exact pathologists, in reference to obscure or unfrequent disorders, but one which when generalized upon as in the present case appears to us to involve as much of assumption as anything that these same generalizers condemn.

A noticeable omission in the discussion of neuritis migrans, which takes up so large a part of the monograph, is that of any mention of the pathological and clinical history of tetanus, which so frequently suggests ascending neuritis. Indeed, we have long been of the opinion that it could have no other pathological process for its basis, and nothing in any recent researches has justified any change of views.

The conclusions of the memoir are stated by the authors as follows:

1. "There exists in certain cases of ascending paralysis an alteration of the anterior roots.

2. "This alteration, which involves a certain number of fibres in each root, is likewise met with in the muscular nerves.

3. "This alteration, analogous to that which we observe in the peripheral end of a divided nerve, is probably of an inflammatory nature (parenchymatous neuritis).

4. "We do not believe that this alteration of the roots is primary, or that it constitutes the sole lesion of ascending paralysis; we believe, rather, that it is consecutive to an alteration of the grey substance of the spinal cord, an alteration as yet inaccessible to our present means of investigation, but which seems probable from the clinical progress of the disease."

M. Gros' memoir is on a somewhat different plan from the other, though treating of the same subject. Instead of a close, critical discussion of pathology, we have a comprehensive paper on a class of cases of which the paralysis of Landry is only one type or termination. Dr. Gros includes under his head of disseminated neuritis, three categories of cases, of which he gives ten observations in full detail, culled from the writings of Dumenil, Lancereaux, Desnos and Pierret, Tripier, Eichhorst and others. The following are his three forms of disseminated neuritis:

I. "An acute form lasting three weeks (obs. IX.), but too rapid to permit muscular atrophy to be clinically demonstrated, and terminating in death.

II. "A sub-acute form lasting from six months to a year, in some instances terminating in recovery, and remaining localized in one point, with more or less complete recuperation of movement, but with no assurance against a new attack (obs. II., III., IV., VI., X), but in other cases terminating in death (obs. VI.).

III. "A chronic form, the most frequent, may continue as long as five years (obs. I., V.). It is in this form that we can now rank obs. VIII., where the disease remaining quiet and without revealing itself otherwise than by a few pains, for a period of thirty-six years, suddenly revived and killed the patient in a few days."

It will be seen from the above that M. Gros' disseminated neuritis includes a large class of cases not exactly corresponding in clinical history with the typical acute ascending paralysis of Landry. Nevertheless, if the species is to be based on the pathological lesions so far as known, rather than on the symptoms, he is right in his classification, as it appears to us.

Of course with so many of these cases having a possibly favorable termination, the question of treatment is of importance. After his chapters on diagnosis, etc., the author ends his memoir with one on the medical and surgical management of the disorder. Besides the measures indicated on general principles, electricity, counter-irritation, etc., he mentions among the appliances that may be employed, hypodermic injections of nitrate of silver and chloroform, and the operation of neurotomy. The memoir, without being very profound or exhaustive, is well worthy of attention.

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IV. The Multum in Parvo Reference and Dose Book. By C. Henri Leonard, M. A., M. D. Third edition. Revised and enlarged. Detroit, 1879.

Pocket Therapeutics and Dose Book, with classification and explanation of Medicines, etc., etc. By Morse Stewart, Jr., B. A., M. D. Second edition, revised and enlarged. Detroit, Mich.: Geo. D. Stewart, 1878.

V. Analysis of the Urine, with Special Reference to the Diseases of the Genito-Urinary Organs. By K. B. Hofmann and R. Ultzmann. Translated by T. Barton Brune, M. D., and H. Holbrook Curtis, Ph. B. New York: D. Appleton & Co., 1879. 269 pages, 8 pl. Chicago: Jansen, McClurg & Co.

VI. A CLINICAL TREATISE ON DISEASES OF THE NERVOUS SYSTEM. By M. Rosenthal. With a Preface by Prof. Charcot. Translated from the Author's Revised and Enlarged Edition, by L. Putzel, M. D. 2 volumes. 1879. New York: Wm. Wood & Co.

I. We have already reviewed the French edition, of which this is a translation, and will only mention such matters as appear worth noticing in this English version.

The translator has taken the liberty of abridging the work; as he says, "In order to bring the subject within the required limits it has been necessary to condense as much as the preservation of the original plan of arrangement would permit, and at the same time to eliminate everything that was not essential to the subject. Hence, perhaps, the most interesting portion of the work, that which dealt particularly with illustrative cases and extended description, was unwillingly sacrificed in order to present the work in its abridged form."

Of the translation we need not say much; it is, for the most part, fairly done, though it must be admitted that it might be improved.

The most objectionable feature, however, is the appending of the other memoir of the author, "Poisons of the Intellect," to the volume. It contributes nothing to the scientific character of the volume, and the recommendation by the author and insertion by the translator causes both to appear at a disadvantage. It would have been much more in place in a popular magazine.

II. This comprehensive little contribution to cerebral anatomy and topography is the latest of Prof. Pansch's valuable publications. He suggests a nomenclature wholly different from his collaborators' and that which he himself has used in past years.

He separates fissures from sulci, and making the first named the more important, confines the latter designation to cortical furrows beginning in the 6th month of fœtal life. His omission of the two sulci inferior to the temporalis superior of Ecker, while retaining the convolutions of which they form the boundaries, does not seem proper. The other authors find these sulci too useful as landmarks to be ignored so completely.

The joining the præcentral sulcus to the inferior frontal, and thus departing from the methods of his companions in this field, we must regard as unfortunate, for two reasons: primarily, it necessitates a change from the system of numbering and designating the lobules in that region, uniformly adopted by anatomists who treat of the subject; secondly, it will be productive of as much confusion as the older want of separation between the occipital and calcarine fissures, merely because in some cases they were confluent.

With such exceptions we cannot but regard Prof. Pansch's little book as a step in the right direction toward a correct classification. Diagnosticians, students of cerebral localization of functions, and pathological anatomists generally should thank this hard-working author for this effort toward a more correct nomenclature. S. V. C.

III. This is one of Wood's dollar series of republished medical works, and one that appears to us to be of considerable value. The American editor has considerably condensed the English original, and has made a number of additions and changes to bring the work into convenient compass, and to render it more serviceable to the American reader. As it comes from his hands we should think it decidedly improved.

This volume only contains accounts of medicinal agents of organic origin, and therefore is far from being a complete handbook of therapeutics, but it seems quite complete so far as it goes. The arrangement is botanical, and therefore all troubles as to therapeutic classification are avoided. Considering the low price and the general merits of the volume, the publishers have done a good work in thus presenting it to the American medical profession.

IV. These two little volumes, both hailing from Detroit, are quite complete in their way. The first contains, besides the usual list of remedies and their doses, a list of pharmacopœial preparations showing their composition, with remarks on the method of their preparations, list of incompatibles, of poisons and their antidotes, together with the principal and most useful chemical tests, and tests for urine examinations, obstetric memoranda, visceral measurements, directions for emergencies, etc., etc. The second, besides many of these, has a list of diseases and their remedies, and a chapter on general symptomatology, which, though more scientific, reminds us much of the family homeopathic manuals so commonly seen in the hands of dosing mothers, which, with their accompanying cases of sugar pellets, have so often relieved the parent's anxiety to do something. It would serve as a handy book for the sailor or traveler who would care to be his own doctor.

It is common to disparage this class of books, and to say that a competent physician has no need of them. We do not share this opinion, and are inclined to think that its motive is not altogether unakin to a sort of sciolism which forms so serious a temptation to many in the profession. Few doctors would care to consult a dose book before their patients, and yet the time frequently occurs when such a proceeding might be advantageous. They are often, as in the present cases, ahead of the pharmacopœia and the usual manuals in mentioning novelties in therapeutics, and while every physician should have a ready knowledge of the standard remedies, there are occasions, and we think they are not infrequent, when the most experienced one might profit from a moment's consultation with a little pocket volume such as these. It must be recollected, that it is not merely the question as to dose, but those in regard to compatibility, solubility, and antidotes, etc., that are answered by such volumes as these. When carefully and properly compiled they can be very useful, and we see no more impropriety in their consultation by physicians than in the use of a pocket dictionary by a student.

Dr. Leonard's work, it is stated, has been carefully proof-read some ten different times by seven different persons, and it seems to be quite free from errors, though we notice some omissions. The other little volume of Dr. Morse Stewart's does not, on the rapid examination we have given it, show any important mistakes.

V. The translators of this work say in their preface "We do not know of a single work in the English language where, in a concise form, so many valuable suggestions and practical hints are offered, both as regards analysis and diagnosis. The book does not pretend to be an elaborate and exhaustive treatise on the diseases of the genito-urinary organs; such an idea is at once precluded by the title and size of the work. It merely claims to contain all that is necessary for the student and practicing physician." While this may be and probably is too unqualified an assertion, it nevertheless appears to be a work of real value, and one which the translators have done a service to their countrymen in translating. That it had already appeared in other languages than the German is a fact that speaks well for its merits.

The translation appears to be creditably done, though we have had no opportunity of comparing it with the original. The translators have added besides the plates that appeared in the original edition still others from Ultzmann and Hofmann's *Atlas of Urinary Sediments*, and the general appearance and make-up of the work is very fine.

VI. In a former issue of this journal (Oct., 1876) we reviewed at some little length Rosenthal's *Klinik der Nervenkrankheiten* in the original, and the favorable opinion of it there given needs only to be referred to here. The publishers have done well to include this work in their cheap series; it is better than a republication of English works dating several years back.

The translation appears to be pretty well done; we should think from some signs that the French edition had been used to some extent in its preparation. It includes Prof. Charcot's introduction to that translation, which is itself a very strong recommendation of the work.

Editorial Department.

IT certainly cannot be considered other than appropriate, that we should congratulate our eminent co-worker—Dr. Hammond—on his restoration to the high rank he had attained in the medical department of the army of the United States, during the early part of the civil war, and on the completeness of his final vindication from the misdemeanors, which it has been since seen, were so unjustly imputed to him.

Dr. Hammond may have even grave faults, as some have thought, but no one familiar with his history during and since the war, will in candor, withhold a sincere admiration for the courage, industry, indomitable perseverance and elasticity of spirit which he has shown, in the midst of a host of difficulties which would have disheartened, or even crushed an ordinary man.

He has triumphed over them, and has placed himself, in many respects, at the head of his profession in his own country, and has made himself widely and favorably known abroad. All this has been accomplished in a comparatively short period in time, and by his personal exertions, apart from outside aid, and even in spite of hostile opposition.

Dr. Hammond has not yet passed his prime, and retains in full measure his physical and mental powers, and lives in the enjoyment of a notable success, both as a practitioner and author, especially in nervous and mental diseases—in that high and difficult department in medicine, which he was the first among American physicians to occupy in a prominent manner.

May he long live to enjoy the well earned rewards of a successful professional life.

We have received the prospectus of a new quarterly neurological journal, "*The Alienist and Neurologist*," to appear about the first of January. It is to be under the editorial and business management of Dr. C. H. Hughes, who is favorably known to very many of our readers as a prominent ex-asylum superintendent and writer on neurological subjects. The scope of the proposed periodical is indicated in its name, but we understand that it will also give brief records of general medical progress, and that its principal aim will be to introduce psychiatric and neurological subjects to the general practitioner. We wish the new journal all the success it merits.

Periscope.

a.—ANATOMY AND PHYSIOLOGY OF THE NERVOUS SYSTEM.

TERMINATIONS OF SENSORY NERVES.—In an inaugural thesis (*Beiträge zur Kenntniss der Endigung der sensiblen Nerven*, Strasburg, 1879), calling for a notice, quite exceptional for that class of literature, Dr. V. Izquierdo communicates his own observations on the above subject, the work having evidently been done in Waldeyer's laboratory.

He prefaces his remarks with an exceedingly full, but not critical, digest of the literature.

The *nerves of the cornea* were examined, in the first place, mainly with Ranvier's mode of gold-staining. According to this method, the freshly excised cornea is placed during five minutes into filtered lemon juice, hereupon immersed, for the space of 20 minutes, in 3 ccm. of a 1-per cent. solution of chloride of gold, and finally kept during 3 to 4 days in 30 ccm. distilled water, containing 2 drops of acetic acid. After hardening in alcohol, sections were made in both directions, or the entire cornea was examined after mechanical removal of the epithelium.

The animals employed were rabbits, pigeons, and canary birds—birds showing especially well the termination of the nerves in the cornea proper. Passing directly to the finer fibres, he describes the formation of the stroma plexus, immediately underneath the anterior lamella in rabbits, but more posterior in birds. At the points of junction of the fibrillæ, cells are found resembling ganglionic cells, but in reality belonging to the connective tissue. At these places there exists also an accumulation of cement, stained red by gold. From this plexus two sets of fibrillæ can be traced, the short *rami perforantes* passing through the anterior lamella, and ending in the epithelium, and a set of longer fibres terminating in the substance proper of the cornea. These longer fibrillæ pursue their course either in separate channels, tunneled through the cornea, or pass along in the canaliculi of the corneal corpuscles, hence bending occasionally at right angles. They rarely remain in one lamella, but pass obliquely forwards or backwards. They *terminate either abruptly with free ends, or connect with the protoplasmic process of the corneal corpuscles.* The latter mode is very well shown in the annexed plates; but the drawings would certainly be more trustworthy were they to represent the entire field of the microscope, and not only, as is stated, nerve-fibril and cell.

The rami perforantes, after passing through Bowman's membrane, form a set of tassel-like tufts—the so-called sub-epithelial plexus. The author agrees with Krause in locating the plexus immediately outside of the "foot plates" of the deepest layer of epithelial cells. In the description of the termination of the fibres, the author confirms Hoyer. The fibrils pass anteriorly up to the most superficial epithelial cells, beneath which they bend at right angles, or nearly so, and pursue a short course in a direction parallel to the surface of the cornea. This last portion of the fibrillæ is not thinner than the rest. The fibrillæ cross each others' paths in many places, but do not anastomose; they are distinctly varicose. *They end abruptly, sometimes with a rounded end, but which is not thicker than an ordinary varicosity.*

The tactile globules in the beak of the duck are the next objects described. We need not follow him in this, since he agrees fully with the description of Hesse.

Studying the development of these bodies, he found the "covering cells" derived from the epithelium, which, when separated from the rest of the epithelium, receive a capsule of connective tissue, while the nerve-fibres advance and finally enter the tactile globule.

In the next place the terminal bulbs (*Endkolben*) in the clitoris of the rabbit are described. The method consisted in immersion of the excised organ in a 3-per cent. solution of acetic acid, for the space of three days, removal of the epithelium with a brush, and hardening in ½ per cent. solution of osmic acid (24 hours), and subsequently in alcohol.

A section through the entire organ reveals a bundle of nerve-fibres, radiating and terminating in the bulbs which surround the entire body of the clitoris, like a wreath. Some of the bulbs are round, but most of them cylindrical, with the axis perpendicular to the surface. Some few bulbs are branched. They consist of a capsule, an inner bulb, and one or two nerve-fibres entering the latter.

The capsule consists of connective tissue in one or more layers. The contents of the capsule—the inner bulb—are either large cells with large nuclei and more or less indistinct outlines, appearing as if their protoplasm had become confluent, or simply a granulated mass without definite structure.

The nerve-fibre (medullated) enters at one pole of the bulb—sometimes a fibre enters at each pole. In the case of branched bulbs, the nerve branches likewise. The sheath of Henle is continuous with the capsule of the bulb, while the medullary sheath disappears gradually, after the entrance of the fibre. Where the sheath of Schwann ends could not be decided. The axis-cylinder terminates either abruptly, or with a point, without further connection with the contents of the bulb.

The last observations refer to the Pacinian corpuscles of the cat. The previous descriptions of the capsule and contents are simply confirmed. The latter is mentioned as a finely granular mass, in the periphery of which nuclei are found. In the midst of this mass the pale nerve-fibre, consisting only of the axis-cylinder, is seen, possessing a distinct striation due to its fibrillar composition. The thickness of the fibre varies; it is usually ribbon-shaped; sometimes it tapers towards the end.

The nerve-fibre, however, is not in immediate contact with the granular contents of the corpuscle, but separated from it by a membrane-like layer of homogeneous, strongly refracting substance. The layer is not of uniform thickness, but shows varicosities, and sometimes fine striæ radiating from it into the surrounding mass. Izquierdo favors the view, that this layer is a delicate continuation of the medullary sheath of the nerve. The nerve-fibre terminates usually near the further end of the corpuscle. In some instances it perforates even the capsule, being enclosed within a tube lined with numerous nuclei, which occasionally makes a bend. The ultimate termination is either in the form of a slight tumefaction, or (more rarely) a free point. This tumefaction is sometimes globular, more often irregular in shape. It seems to be a thickening of the substance of the fibre itself.

H. G.

NERVI ERIGENTES.—According to Eckhard and Loven, the nervi erigentes arise from the first and second (rarely third) dorsal roots, passing into the hypogastric plexus, and thence with the branches from the plexus into the walls of the vessels of the penis.

Nikolsky (*Arch. fuer Anat. u. Phys.*, 1879, III. and IV. Heft, p. 209) adds to this description that the anterior or upper root of each erector nerve (the branch from the first dorsal root) is thinner than the posterior or lower root (coming from the second dorsal nerve), and that it alone receives a twig from the ramus communicans of the first sacral sympathetic ganglion. In the course of the nervi erigentes nerve-cells are found, on the posterior and lateral surface of the pars membranacea urethræ, in its connective tissue sheath, and along the fibres passing from the hypogastric plexus to the membranous portion.

The function of the nerves was tested by electric irritation, measuring at the same time the quantity of blood flowing from a divided dorsal vein of the penis. It was found that irritation of the posterior root alone of the nervi erigentes produced vascular dilatation and erection of the penis; section of these fibres allowed the vessels of the penis to contract under the tonic influence of the vaso-constrictor nerves. The anterior thin root on the other hand proved to be vaso-constrictor in its function, counteracting the effect of the dilator fibres of the nervus erigens. The pudic nerve possesses also a constricting influence upon the vessels of the penis.

The dilating fibres of the nervi erigentes are paralyzed by atropia, but stimulated to action by muscarin and asphyxia.

THE POINT OF ENTRANCE OF NERVE TRUNKS INTO MUSCLES.—In searching for a principle determining the entrance of nerve trunks into the body of the muscles, Schwalbe (*Archiv f. Anat. und Phys.*, 1879, III. and IV., p. 167) has discovered the following points:

The point of entrance of the nerve depends on the shape of the muscle, which determines also the site of the motor terminal plates. Kühne has found that the latter are situated symmetrically to a certain distance on each side of the centre, in muscles consisting of parallel longitudinal fibres. In such muscles, if about equal in breadth and thickness, the nerve enters in the centre of the belly of the muscle, for instance, teres major and minor, tensor fasciæ latæ, and some ocular muscles.

If the muscles are very long, like the sartorius, several nerve branches enter arranged in a "nerve-line" which runs parallel with the direction of the muscle fibres, and extends to a symmetric distance on each side of the centre of the muscle. Muscles, the breadth of which exceed 2 to 3 cm., are also supplied by several branches arranged in a nerve-line vertical to the length of the muscle, for instance, gluteus maximus.

In triangular muscles the point of nerve entrance approaches the tendon towards which the muscle-fibres converge, its proximity increasing with the convergence of the fibres, no matter whether this tendon be the central or distal end of the muscle. Since such muscles measure usually more than 2 to 3 ccm. in breadth at their broad insertion, they are supplied by several nerve-branches in the shape of a nerve-line. In spindle-shaped muscles the point of nerve-entrance is again situated in the centre.

All these variations are explicable on the principle *that the nerve enters the muscle in the geometric centre of the latter*. This principle is illustrated by Schwalbe by a series of mathematical considerations, for which we must refer the reader to the original.

THE INFLUENCE OF EXCITATIONS OF PERIPHERAL NERVES ON THE NERVE CENTRES.—M. Onimus, *L'Union Médicale*, July 29, had his attention called to the fact, that in numerous cases in which he had for various disorders applied the galvanic current to the sciatic, there followed the electrization a profound sleep. That this was not due to the relief of pain by the operation, is probable from the fact, that in many of the cases sleep was not ordinarily impaired from any such cause. The sleep produced by the galvanization of the sciatic was a profound, heavy slumber, and waking at the usual hour was even difficult. M. Onimus considers this effect to be due to a direct action on the cerebral circulation, and in support of this view, among other clinical and physiological proofs, he adduces the experiment of Brown-Sequard, who rendered rabbits epileptic by the section of the sciatic nerve.

Although he does not consider electrization of the sciatic as an always efficient means of producing slumber, he thinks the fact is suggestive in many points of view, clinical and therapeutical. He calls especial attention to the importance of protecting the peripheral nerves from chill, alludes to the fashionable but unphysiological practices in dressing children, leaving the lower limbs largely exposed, or but poorly protected, and states that in all the cases of infantile paralysis in regard to which he had been able to obtain accurate data, the cause was cold in some form or other, and in some it was directly attributable to chilling the lower limbs. While the cerebral and spinal centres may be affected in a reflex way through all the peripheral nerves, the sciatic appears, of all others, to exert the greatest influence in this manner. Reciprocally it is in the sphere of the sciatic that the eccentric irradiations from the spinal centres are most acutely felt, and even in cerebral affections this nerve may be similarly involved. The paper, though short, is eminently practical and valuable.

THE SUPERIOR MAXILLARY NERVE AS A TYPICAL VASO-MOTOR.—At the session of the Soc. de Biologie, June 28, M. Laffont read in his own name, and that of M. Jolyet, a paper on this subject, which is thus reported in the *Gaz. des Hopitaux.*

At a preceding session these experimenters had announced that there are in the superior maxillary vaso-dilator nerve fibres, properly so-called, acting on the labial, nasal and gingival mucous membranes.

In a new series of researches they studied the phenomena in the triple point of view as regards heat, redness and vascular pressure, which may be considered as the tripod of vascular dilatation, and in fact the results have always agreed, no matter in what way the phenomena were studied.

1. In all the experiments in which the authors studied the effects of the electrization of the peripheral end of the superior maxillary nerve on the temperature (the temperature being taken simultaneously in the two nostrils by means of very sensitive thermometers), they found an elevation of between one and four degrees centigrade on the side operated on.

2. As regards the reddening, all the experiments have also shown that electrization of the peripheral end of the superior maxillary nerve causes an intense redness of the nasal, labial and gingival mucous membranes of the same side. The lip swells, the tactile hairs bristle on account of the turgescence of the skin, and appear like umbilici. All these phenomena are the better observed when we operate on animals in which the lips are unpigmented.

3. The tracing shown by M. Laffont to the Society, and which represented the vascular pressure taken in continuity in the maxillary artery by means of a T arrangement, shows that in this, as in the lingual artery during electrization of the peripheral end of the lingual nerve, as MM. Jolyet and Laffont first demonstrated in Nov., 1878, *the primary, immediate and persistent effect is a reduction of the arterial pressure.*

The investigations of these gentlemen therefore put us in possession of a third typical vaso-dilator nerve, and their discovery takes rank with those of Cl. Bernard on the circulation of the sub-maxillary gland, and that of M. Vulpian on that of the tongue.

Moreover, after this discovery, we should reject the hitherto dominant theory for the explanation of emotional congestions and congestions accompanying trigeminal neuralgias, which admits in these cases a temporary paralysis of the bulbar vaso-motor centres.

Finally, we have now a new proof of the existence of peripheral vaso-motor centres, as we can, as MM. Jolyet and Laffont have already said in 1878, explain this primary and immediate dilatation only by an action of these vaso-dilator nerves (for which the centre is in the medulla or brain) on the peripheral vaso-motor centres, whence they come temporarily to suspend, when they are excited, the tonic and permanent action of the vaso-constrictors.

MM. Jolyet and Laffont have, moreover, produced an experimental proof of the fact first announced by M. Cl. Bernard, that the dilators obey a less energetic excitant than do the constrictors. Indeed, when they had electrized the cervical sympathetic with a very strong current, they have seen on exciting the peripheral portion of the superior maxillary with a very weak current, a maximal pallor suddenly follow an intense redness.

These vaso-dilator filaments arise, according to the investigators, from the vidian nerve, the electrization of which gives the same effects as electrization of the superior maxillary nerve.

ORIGIN OF THE MOTOR NERVES OF THE EYES.—At the session of the Société de Biologie, June 28 (rep. in *Gaz. des Hopitaux*), M. Math. Duval reported the following results of his investigations as to the origin of the ocular motor nerves:

The nuclei of the origin of the third, fourth and sixth pairs of cranial nerves are connected together by bundles of fibres, the arrangement of which explains the complicated physiology of the associated ocular movements. Previous to the experimental researches of M. Laborde and the clinical facts published by M. Fercol and M. Graux, he had ascertained, in great measure, the anatomical facts now stated, and had recently completed the study by means of sections of the medullas of apes. A very marked bundle goes from the nucleus of the third pair to that of the sixth of the opposite side; thus we can understand the simultaneous contraction of the right internal rectus and the left external rectus, and inversely. This bundle is double in the ape, one of its branches enters the nucleus of the patheticus. This nerve, therefore, takes its origin partly from the muscles of the external motor oculi; hence there is nothing astonishing that it innervates a muscle rotating outwards, such as the superior oblique. In movements of lateral inclination of the head, this nerve acts simultaneously with that of the inferior oblique of the opposite side, but in an inverse sense. But the nucleus of the patheticus blends itself with the posterior part of that of the third pair, the point of origin of the motor-oculi communis, which innervates the inferior oblique, and the patheticus itself, decussating in the valvule of Vieussens, passes to the superior oblique of the side opposite to that on which it seems to rise. The simultaneity of action of the two oblique muscles in question is thus explained by the community of origin of their different nerves.

PHYSIOLOGY OF THE CIRCLE OF VIEUSSENS.—At the session of the Soc. de Biologie, July 10 (rep. in *Le Progrès Médical*), M. Francois Franck reported that he had made numerous experiments on animals to determine what share of iridian, carotidian, vaso-motor, and cardiac accelerator fibres are contained in the two branches of the circle of Vieussens, which unites the first thoracic ganglion to the cervical cord of the great sympathetic. In a curarized animal he tied the anterior branch of the circle of Vieussens; there followed a dilatation of the iris, bilateral and temporary, proving that it was a reflex phenomenon. If, on the other hand, instead of tying this branch, we divide it in the middle, the excitation of its peripheral portion causes dilatation of the pupil of the same side, while excitation of the central portion causes dilatation of both pupils. There are no pupil-dilating fibres in the posterior branch of the circle of Vieussens. As regards vasomotor fibres, they are distributed in apparently the same proportion in the anterior and posterior branches of the circle of Vieussens.

The following are the titles of some of the recently published articles on the Anatomy and Physiology of the Nervous System :

Pawlow, On the Innervation of the Blood Routes, *Pflueger's Archiv*, XX., 4 and 5 Apr.—Vintschgau, On the Physiology of the Sense of Taste, *Ibid.*—Poole, Effects of "Pithing" on the Vascular System, *N. Y. Med. Record*, Sept. 13.—Lewinski, On the Power Sense, *Virchow's Arch.*, XXVII., 134.—Luys, Studies on the Duplication of Cerebral Operations, and on the Separate Rôle of each Hemisphere in Mental Pathology, *L'Union Med.* (cont. art.)—Maragliano, Experimental and Clinical Researches on the Cerebral Temperature, *Rivista Clinica*, July and Aug.—Tschiriew and DeWatteville, On the Electric Excitability of the Skin, *Brain*, July.—Galton, Psychometric Experiments, *Ibid.*

b.—PATHOLOGY OF THE NERVOUS SYSTEM AND MIND; AND PATHOLOGICAL ANATOMY.

Tender Spinal Points in Visceral Affections.—At a meeting of the Soc. de Biologie, June 28 (rep. in *Gaz. des Hopitaux*), M. Vidal reported as the result of his observations that certain visceral affections are accompanied with pain in certain parts of the vertebral column, and that these sensitive points depend, as regards their situation, upon the organ affected. In ulcer and cancer of the stomach, pain is felt at the horizon of the sixth dorsal apophysis; in hepatic affections, at the fourth; and finally, in perityphlitis, especially if the inflammation affects the cellular tissue of the iliac fossa rather than the peritoneum, the pain is felt at the points of emergence of the two first left lumbar pairs.

M. Leven remarked that in stomach diseases the pains should be distinguished as to whether they were due to the pneumogastric or to the grand sympathetic. In the first case they coincided with the dyspnœa, the sensations of suffocation and palpitations, in the second they are more deeply situated, and are accompanied with vaso-motor disorders of the left arm and the corresponding half of the trunk.

M. Laborde disagreed with M. Vidal as to the existence of special visceral centres at definite horizons in the cord, corresponding to apophysary tender points; he combated the interpretation of the facts, not the facts themselves.

Laryngeal Chorea.—A most interesting article on this subject, by Prof. Schrötler, of Vienna, was published in the *Allg. Wien. Med. Zeitung*, No. 7, 1879. The affection, as described by Schrötler and others who had written on the subject previously, presents a very striking and complex group of symptoms, exceedingly annoying and trying, not only to the patient but to those around. The prominent symptom of the disease is a cough over which the patient has no control, and which is totally different from that observed in other affections of the air passages. The cough may be either of a barking, howling (heulenden) or crowing character. In certain cases of hysteria such a cough may occur, and it is occasionally simulated, but in many cases it cannot be traced to either of these causes. Bell observed, in a girl fifteen years old, a convulsive, barking cough, which occurred ten times per minute, and disappeared during sleep. It did not lead to any disease of the larynx. The disease lasted four weeks, and relapsed three times.

Romberg describes, under convulsive affections of the "nerves of voice," an abnormal tone of voice, which he thought was due to an "hysterical or epileptic condition." He reports the case of a young woman nineteen years old, who made a sound "like a saw-mill," and so loud that it could be heard on the steps outside the house. He also mentions the case of a physician, sixty years old, who, in consequence of a complicated nervous affection, had occasional attacks of loud bellowing coughing. A singular feature in this case was, that soothing influences, and especially music, had the power of preventing the paroxysms and lessening their violence.

Mandl described similar affections. More recently Türck describes similar cases under the head of "convulsive coughs," but Schrötler thinks it doubtful whether they were due, as Türck supposed, to disturbances of innervation about the larynx. In these cases irresistible paroxysms of coughing occurred, which were of short duration, and of a peculiar barking character. The larynx, trachea and bronchi were perfectly healthy. Türck reports five cases—four of which presented precisely the symptoms of those observed by Schrötler. Massei has reported three cases, which he thought due to hyperæsthesia. His patients were aged respectively, twenty-four, fourteen, and eighteen years, and they presented very slight or no laryngeal disease which could be detected. He considers the affection often incurable. Geissler reports the case of a boy, twelve years old, who, after taking cold, had the characteristic cough, which was brought on whenever he attempted to pronounce words or syllables commencing with the letter H. Subsequently he suffered with general convulsions, with hallucinations and subsequent coma, attacks of which could be brought on by pressure or pinching of certain points. Prof. Wagner, in Leipsic, thought the affection in this case a form of hysteria. Dr. Spamer, in Giessen, has reported a case of the peculiar cough occurring in a child a year and a half old, after an attack of general chorea. Schrötler has had eleven cases. They were all young persons, generally from eight to fourteen years of age, in whom, without known exciting cause or any other disease, attacks of coughing frequently occurred. The attacks came on every five or ten minutes when the patient was awake, and disappeared during sleep. He proposes to call the affection laryngeal chorea—chorea, because the spasmodic contraction of certain muscles or groups of muscles occurs during the waking movements of the patient, and disappears during sleep; furthermore, the patient has no control over the attacks; laryngeal chorea, because the most prominent symptoms have their seat in the larynx. Other groups of muscles in no way connected with phonation and respiration are occasionally affected. That the affection is a form of chorea is rendered further probable by the mimicry, the relapses, of course, of the disease, and the occurrence of other nervous affections in the same individual, or in the members of the family.

Of the eleven cases seen by Schrötler, there were three boys and eight girls—one of the latter being twenty-one years old. All were of delicate constitution; two were anæmic. In some there was a very slight catarrh of the larynx or trachea present; in others this was absent. Schrötler thinks the affection a motor neurosis in the strictest sense of the term; but whether it has its origin in the central nervous system he considers a matter of speculation only. The prognosis is favorable—all of his cases having recovered in from three to six weeks. Relapses occurred, but they were always less severe than the original disease.

The treatment consisted in cold shower-baths two or three times a day, the administration of quinine in large doses, and the application of the constant current. Iron was given to the anæmic patients.—*Virginia Medical Monthly.*

ATHETOSIS.—W. Murrell, M. R. C. P., reports (*London Lancet*, Am. rep., July) the post-mortem appearances in the brain of a man aged 33, who had had athetosis of the left hand, following convulsions and paralysis at the age of three years. There had been little change in the symptoms for twenty years, there was no pain, loss of sensation, nor fatigue. The movements were confined to the left hand, except on rare occasions after great fatigue, when something similar was observed in the left leg. There was considerable wasting of the muscle of the left arm, especially on its posterior surface. The cause of death was phthisis, the immediate cause being diarrhœa and exhaustion. The following are the results of the autopsy, made thirty-six hours after death, in the presence of Drs. Sturges, Allchin, Gowers, and others:

"Rigor mortis marked; no post-mortem congestion; great emaciation and pallor of skin; calvaria rather under than over normal thickness; no local thickenings; dura mater not more than ordinarily adherent; halves of skull symmetrical; dura mater of normal appearance externally; no grittiness of sinuses or vessels; large veins over posterior part of pia mater distended by partially coagulated blood; blood in vessels contained considerable quantity of air-bubbles; Pacinian bodies numerous along posterior part of longitudinal fissure, none in front; no undue thickening of meninges; from three to four drachms of clear serum escaped on removing dura mater. Brain: The whole right hemisphere distinctly smaller than the left, about three-quarters of an inch shorter; the two parietal lobes, viewed from above, were nearly equal, but the frontal lobe on the right side was considerably narrower than on the left; the posterior half of the middle and inferior frontal convolutions, and to a slighter extent the superior frontal and ascending frontal, were distinctly smaller on the right side than on the left. There was a distinct difference also between the anterior portions of the frontal convolutions on the two sides, although this was slighter than in the posterior portions. The occipital convolutions on the two sides were nearly equal, but those of the parietal lobes were distinctly smaller on the right than on the left.

"There was a deep depression extending backwards into the lobe, about three-quarters of an inch deep, and bounded by somewhat atrophied convolutions, on the surface of which some of the white substance of the brain appeared. No difference in size was to be observed between the two halves of the cerebellum ; the orbital lobule, like the rest of the right hemisphere, was a little smaller than the left ; the right anterior pyramid was very conspicuously smaller than the left. The arteries at the base appeared to be perfectly healthy. Further examination showed that the convolutions of the island of Reil were apparently normal, but there existed on the inner side a deep excavation between the anterior extremity of the perforated spot and the convolutions, extending backwards between the convolutions of the island of Reil and operculum as far as the anterior surface of the hemisphere. Close to the anterior surface of the hemisphere the fissure was three-quarters of an inch deep, and its total length appeared to have been two inches and a half. The sides seemed to have been in apposition, except outside the perforated spot, where the cavity was about a quarter of an inch wide ; and its roof was formed by radiating fibres spreading upwards from the pons—probably those of the external capsule. On the inner edge of this fissure the outer roof of the olfactory nerve was seen crossing backwards to the temporo-sphenoidal fissure. Ventricles opened : On examination, almost the whole of the right corpus striatum appeared to be destroyed—at least that portion in front of the optic thalamus. The posterior portion of the nucleus caudatus outside the optic thalamus was unaffected. A small portion of the inner part of the corpus striatum near the middle also appeared intact, but the whole of the grey substance was destroyed, so that there was only a membranous septum separating the cavity of the ventricle from the deep fissure which has been described. This septum consisted of a double membrane, and in it were seen a number of strands of white fibres, apparently belonging to the internal capsule. The optic thalamus seemed to be quite healthy; no naked-eye change detected in the cord."

EARLY SYPHILITIC AFFECTIONS OF THE NERVOUS CENTRES.—Prof. Mauriac closes a long and able paper on this subject, with the following deductions from the facts and researches at his command:

1. Syphilis may attack the nervous centres at a very early period after the initial lesion.

2. The early cerebro-spinal lesions are those which develop during the virulent period of the malady—that is to say, during the first two or three years.

3. There are degrees in this precocity of the cerebro-spinal syphiloses: the first include those which set in within the first twelve months; the second, those which develop in the second or third year of the constitutional malady. Statistics seem to show that those of the first degree are more common than those of the second.

4. Among the early visceral localizations of syphilis, those in the cerebrospinal system are incomparably the most numerous.

5. They are also the most dangerous. Their gravity does not increase with their diathetic age; those which develop during the first months of syphilis are as formidable as those which belong to the more advanced stages of the malady.

6. All the forms, all the degrees, all the phenomenal combinations that constitute the symptomatology and the processus of the localizations of syphilis in the neural system are met with in the early as well as in the late cerebro-spinal syphiloses.

7. Certain symptomatic complexes, however, seem to predominate. The most frequent are those which consist in an attack of hemiplegia, involving the whole of one side of the body.

8. Among the attacks of hemiplegia, the syndroma, comprising right hemiplegia and aphasia, is the most common.

9. The paralytic forms are much more common than the convulsive or epileptic in the early cerebral syphiloses.

10. In the cerebro-spinal syphiloses the psychical troubles and the incoordination of movements are never systematized as they are in mania, general paralysis and locomotor ataxia.

11. The absence of systematization in the cerebro-spinal syphiloses must be regarded as one of their chief characteristics. The only exception is in the case of the syndroma of right hemiplegia and aphasia.

12. Early localizations of syphilis in the spinal cord are much less common than in the encephalon.

13. The lesions which seem to belong to the early cerebro-spinal syphiloses are diffused or, more frequently, circumscribed hyperplastic effusions into the cortical layer of the brain and the pia mater, and changes in the sylvian arteries with consecutive ischæmic softening.

14. In some cases of early cerebro-spinal syphiloses that terminated fatally, no lesion was found, but at that time the existence of arterial syphilis had not been recognized. It may be presumed that death had resulted from sudden anæmia of the nervous centres that are essential to life.

15. With regard to the etiology of the early cerebro-spinal syphiloses, only very vague conjectures can be advanced. In most of the cases the initial lesion as well as the consecutive cutaneous and mucous manifestations were very mild in character.

16. The general cause of the constitutional malady is not modified by the appearance of early localizations in the nervous centres. The other manifestations develop before, during and after the localization in the neural system, without presenting any deviations from their usual forms, degrees, course or topography.

17. The precocity of the cerebro-spinal syphiloses furnishes no special indication with regard to treatment. Whatever may be the age of the constitutional malady, the localization in the nervous centres demand the same specific medication. The peculiar conditions of each case furnish the secondary indications relative to the choice, doses and combinations of the two specific agents.—*Annales de Dermatol. et de Syphilig.*, Vol. X., No. 3.

DELIRIUM TREMENS.—Dr. William J. Scott, of Cleveland, Ohio, asks, *Toledo Medical and Surgical Journal*, July, for observations on the frequency of delirium tremens and insanity among negroes. He had a case of surgical injury some years since in an intoxicated negro, which was followed by tetanus; and during his care of this case, he learned from his patient and friends that they commonly believed themselves free from liability to acute alcoholism of this form. Since then he has directed his observation and inquiries to this point, and has not found any counter-evidence, either from authorities of workhouses, hospitals or from physicians of large experience.

As regards the question as to the occurrence of insanity among the blacks, the question can be very easily answered: it does occur, but with what relative frequency we cannot say; and if delirium tremens does not affect the colored race, it is, to say the least, an interesting fact.

CURABILITY OF INSANITY.—At the meeting of the College of Physicians of Philadelphia, May 7 (rep. in *Phil. Med. Times*, Aug. 2), Dr. Isaac Ray read a paper upon the subject of recoveries from mental disease. A wide difference of opinion has always existed respecting the curability of insanity, which, contrary to expectation, the information gained from the establishment of hospitals in recent times has failed to reconcile. Dr. Earle, in examining the matter of *recoveries* as exhibited in the statistics of hospitals for the insane, has found that thirty or forty years ago the proportion of recoveries was much larger than it has been of late years. Dr. Earle accounts for this by suggesting two sources of error committed by the reporters: One of these depends upon the idiosyncrasy of the individual temperament, constitutional organization, etc. Self-interest and ambition may have prompted more favorable returns in some cases than in others.

Again, Dr. Earle says that "the reported recoveries from insanity are increased to an important extent by repeated recoveries from the periodical or recurrent form of the disease in the same person; and consequently the recoveries of persons are much less numerous than the recoveries of cases or patients; it is generally impossible to ascertain the number of persons who recovered."

These views are not borne out by the facts, properly interpreted. The temperament of physicians, taken together, is just about the same now as it was fifty years ago. There is the same proportion of sanguine men who take too hopeful views of their patient's condition, and cautious men who tend to give unfavorable prognoses. Then, too, the practice of reporting cases of recovery instead of persons permanently cured, was no more common than now.

As, then, neither the temperament of the physician nor the repeated counting of periodical cases accounts for the larger proportion of recoveries in the earlier times; we must look for the explanation in another direction, and we shall find it in various agencies that have come into operation in later times.

These are the ingathering of a larger proportion of non-violent, chronic, less curable cases, dependent upon the wider popularity of hospitals for the insane; the actual increase of insanity in our midst, owing to the high pressure of modern life; and, as a result of this, the appearance of new forms of mental disease heretofore unknown.

These, then, are the points which are believed to have been fairly made, viz.:

I. Those qualities of temperament which lead men to unduly magnify their achievements are as common at one time as at another.

II. The practice of reporting cases instead of persons has not been confined to any particular period; and, therefore, while it may vitiate our estimate of the curability of insanity, it cannot make the proportion of recoveries larger or smaller at one period than at another.

III. Cases marked by high excitement entered our hospitals in a larger proportion to those of an opposite character fifty years ago than they do now.

IV. Under the influence of highly civilized life, the conservative powers of the constitution have somewhat depreciated, and to that extent impaired the curability of insanity.

V. During the last fifty years, cerebral affections in which insanity is only an incident, have been steadily increasing, and thus diminishing the proportion of recoveries.

THE CONJUGATE DEVIATION OF THE HEAD AND EYES.—Grasset (rep. from *Montpellier Médical*, abstr. in *Centralbl. f. Nervenheilk.*, Aug. 1).

Grasset has briefly, in a communication to the Academy of Montpellier (Session, May 5, 1879), on a basis of his own, and the revision of those previously published, reopened the question as to the direction of the deviation of the eyes and head in lesions of the brain. As is well known, Prevost, in 1868, had stated from experiments on animals by himself, and an analysis of fifty-eight cases, that the deviation was *towards the injured side of the brain.* Later, in 1875, Lepine corroborated this statement, while in the following year Landouzy, on the other hand, combated it, having found ten exceptions in thirty-three cases. Grasset, in his five observations, finds two in accordance with Prevost's statement, two others in opposition, while the fifth, in which the lesion was in the pons, could not be adduced in its support; and he therefore undertook to investigate the matter anew. The result, however, reached by him, is directly opposed to that obtained by Landouzy. The latter had remarked very correctly, that we should make a distinction between paralytic and irritative conditions, but then says that the head and eyes are turned toward the paralyzed side, if paralysis is present, but toward the side of the cerebral lesion, and consequently toward the unparalyzed side of the body, if convulsive phenomena affect the latter. Grasset states the direct opposite of this, and demonstrates the correctness of his views by the analysis of one hundred and seventeen cases. (A simple reflection will suffice to show that Grasset's view is the only one according with physiological experiment. In order to ascertain the position of the eyes it is only needful to recall the action of the abducens of the diseased side: with convulsive phenomena this must draw toward the side of the convulsed members, and the eyes would turn from the side of the cerebral lesion, while in paralysis, the reverse would take place. But, sometimes, paralysis and convulsions may occur simultaneously on the same side, and such cases have produced the confusion among authors.—*German Abstr.*)

Five of the one hundred and seventeen cases, Grasset does include as true cases, since in them there was no paralysis of the extremities; eight more are unsatisfactory, since neither head and eyes were drawn to the same side, or the paralysis of the extremities was crossed, or there were other anomalies. Of the remaining one hundred and four cases, ninety-nine were in full accordance with Grasset's law, and of the remaining five only one was an absolute exception.

Grasset next investigated the question of the exact anatomical seat of the lesion that produces rotation of the head and eyes. According to Ferrier, experimental irritation only of the tracts of the convolutions numbered in his scheme 12, 13, 13^1, and 14, will produce rotation of the head, that is, the region at the base of the second frontal convolution, the region above the Sylvian fissure, the two posterior thirds of the first temporal convolution, and the angular gyrus. But later Ferrier (and also Boyer in a recent thesis) has limited the centre for the combined movements of the head and eyes to the foot of the second frontal convolution, and considers the others as only centres of vision. But according to Grasset, the region whence this phenomenon is produced, is much more extensive, and corresponds more nearly to the regions first designated by Ferrier, extending, moreover, down to the pons and the medulla. Of the one hundred and seventeen cases analyzed by Grasset, in sixteen the regions 13, 13^1, and 14, of Ferrier, were involved, *without* implication of the second frontal; in six, the vicinity of these was affected; in ten, these and the frontal; in fourteen the lesion was in the white bundles of fibres passing down from these convolutions; in twenty-five, the internal capsule and the peduncles; in three, the under portion of the pons, the cerebellum, and the medulla; while forty-one cases were unavailable for purposes of localization (from diffusion of morbid process, insufficient record, no autopsy, etc.), and two were altogether exceptional (lesions of second and third frontal, obliteration of art. fossæ Sylvii, softening of anterior central convolution). Grasset therefore considers that the symptoms may be due sometimes to a cortical lesion of the region around the Sylvian fossa, and the gyrus angulatus.

When this paper was offered, M. Landouzy showed the author a paper, already read before the Anatomical Society and shortly to be published, in which he takes back his former conclusions, and, in effect, adopts the same views as Grasset.

CARDIAC NEURITIS.—Attention has recently been called by some continental physicians to a pathological condition of considerable clinical interest and importance, especially in connection with some forms of angina pectoris. Dr. Peter, the eminent physician of La Pitié, and well known as the editor of the last edition of Trousseau's *Lectures*, has been especially concerned in endeavoring to give prominence to the theory that, when symptoms of angina pectoris accompany disease of the coats of the aorta (aortitis) with dilatation of that vessel, the peculiar clinical phenomena observed are due to an inflammation of the nerves of the cardiac plexus; an inflammation which extends from the arterial tunics to the branches of the cardiac plexus in contact therewith. Several cases have been reported and analyzed in support of this view. In one case observed and reported by

Dr. Bozey, the cardiac plexus was carefully dissected at the necropsy, and the nerves composing it were found thickened and presenting moniliform swellings along their course. It has been noticed that in these cases, in contradistinction to others which are purely neuralgic, the pain is permanent instead of intermittent, or at any rate, there are certain points which remain always painful on compression with the finger, especially for example, the intercostal spaces along the left border of the sternum. In some of these cases, compression of the left vagus in the neck would at once induce pain in the region of the cardiac plexus, and the pain would radiate along the course of the branches of the pneumogastric; as, for instance, along the back of the sternum, in the stomach, which becomes distended with gas, and in the region of the pulmonary plexus. M. Bucquoy has also noticed, at the termination of such attacks, severe pain in the hepatic region, which he refers to a participation in the disturbances of the terminal ramifications of the vagus on the right side.

M. Peter maintains that the aortic insufficiency which frequently co-exists in such cases, must be regarded simply as a "contingent fact," and as quite incapable of itself to excite the paroxysms of angina. He, however, is especially urgent in calling attention to the far more serious significance of aortic insufficiency when it arises from an affection of the arteries, when disease of the aorta extends to and implicates the sigmoid valves, than of aortic insufficiency of endo-cardial origin; since the former condition commonly leads to a cardiac neuritis, with all the grave phenomena of angina pectoris. M. Huchard has also insisted on the fact that the gastro-hepatic disturbances which he has observed in several cases of cardiac disease is exclusively dependent on a phlegmasia of the cardiac plexus; such as severe pain after food, enormous distension of the stomach with gas, pyrosis, anorexia, and vomiting. Hence in cardiac neuritis we may encounter a great variety of symptoms, according to which of the numerous ramifications of the pneumogastric are especially involved; disturbance of cardiac rhythm and syncope, if the cardiac branches themselves be chiefly involved; or gastro-hepatic symptoms or pulmonary symptoms, if the branches going to the digestive or respiratory organs be mainly implicated.

The morbid anatomy of the nerves of the cardiac plexus has been specially examined by Putiatin, of St. Petersburg, who has found in some cases, after death from cardiac paralysis, in which no coarse changes in the cardiac structures could be discovered, a diseased condition of the cardiac ganglia limited to distension of their vessels, and an intermixture of granulation cells with the nerve-fibres and corpuscles.

In older cases, where organic disease of the heart also existed, more decided pathological changes were observed, consisting chiefly in increase of interstitial connective tissue, and atrophy and granulation of nerve cells.

This is a somewhat neglected branch of cardiac pathology, to which we would especially direct the attention of our many able and industrious morbid anatomists.—*British Medical Journal*, July 12, 1879.

INEBRIETY AS A DISEASE.—Dr. T. D. Crothers, *Med. and Surg. Rep.*, July 19, gives the history of three cases, which are selected on account of their fullness and accuracy as to detail, of inebriety, in which the preceding symptoms and heredity of unstable nervous organization were carefully and thoroughly collected. It is, like other clinical articles of the same character by the author, strongly in favor of the pathological theory that inebriety is a disease.

In two of the cases reported there was a history of ancestral drunkenness, and the brother of one was insane; in the third both parents died early of consumption. All three were, as young men, eccentric, two of them were morbidly religious, and all were at one time especially particular as to their habits, mode of life, diet, etc. Two were active in the temperance reform just before they contracted their inebriate habits. All were neurasthenic. Dr. Crothers says: "These three cases bring out prominently the peculiar class of symptoms which I am confident both precede and follow many cases of inebriety. Neurasthenia, with both functional and organic nerve disturbances, is present in nearly all the cases. They all follow in the line of many of the complex physical symptoms which are so puzzling to the ordinary observer, and indicate, without doubt, the disorder of some brain centres or some general condition of debility.

"The presence of these early symptoms is not always followed by inebriety, but they indicate a condition that lies closely along the border-land of this disorder: a condition which, with a slight change of circumstances, develops inebriety at once: all the soil is ready, only needing the germ to spring into activity.

"These cases can be studied in nearly every community, and are seen often in the enthusiastic temperance men, who have a half consciousness of their danger, and hence are more active to help others, and by this means save themselves. These symptoms are of grave importance when developed among brain workers, because, all things considered, less power of resistance and capacity for recovery exists.

"Delusions and morbid convictions and introspections of the mind impress the brain more permanently in some cases than in others, and their presence in any case is a hint of much pathological importance.

"These clinical facts may be condensed into the following statement:

1. "Extreme solicitude for the care and protection of the body, as well as the food and fluids used, when it becomes a delusion, absorbing the thoughts and energies, is, as a rule, an early symptom of inebriety.

2. "Delusions of fear, faith, and extreme anxiety for the temperance reform, in persons not intemperate before, accompanied with changing emotions and unstable nerve conditions, are significant symptoms, very often ending in inebriety."

THE following are a few of the recent articles on the Pathology of the Nervous System and Mind, and Pathological Anatomy:

PUTZEL, Cerebral Complications of Chorea, *N. Y. Med. Rec.*, Sept. 6.—BEARD, Nervous Diseases connected with the Male Genital Function, *Ibid*, Sept. 27.—ERLENMEYER, Suicide in Prussia, *Centralbl. f. Nervenheilk.*, Sept. 1.—BECHTEREW, The Alterations of Temperature in the Insane, *St. Petersb. Med. Wochenschr.*, Aug. 23.—RINGER, Notes of a Post-Mortem Examination on a Case of Athetosis, *Practitioner*, Sept.—Beard, Neurasthenia as a Cause of Inebriety, *Quarterly Jour. of Inebriety*, Sept.—Barton, Dipsomania; its Medical and Legal Aspects, *Ibid.*—Norris, Optic Neuritis as a Symptom of Intra-cranial Disease, *Phil. Med. Times*, Aug. 30.—Caldwell, Hydrophobia, *Southern Clinic*, Sept.—Regis, Dynamic or Functional Exaltation at the Commencement of General Paresis. *Ann. Med. Psych.*, July.—Ferrier, Vomiting in Connection with Cerebral Disease, *Brain*, July.—Hughlings-Jackson, Affections of Speech from Disease of the Brain, *Ibid.* —Stewart, The Eye-Symptoms in Locomotor Ataxia, *Ibid*—Dowse, Remarks on Bulbar Paralysis, with Special Reference to Artificial Feeding, *Ibid.*—Kraemer, Observations of Temperature in the Paralytic Insane, *Allg. Zeitschr. f. Psych.*, XXXVI., I. and II.—Roller, The Pathology of (Precordial) Pain, *Ibid.*—Fröhlich, On Psychoses among Soldiers, *Ibid.*—Pfleger, Observations of Crumpling and Sclerosis of the Ammonis horn in Epilepsy, *Ibid.*

c.—THERAPEUTICS OF THE NERVOUS SYSTEM AND MIND.

Oxalate of Cerium in Pertussis.—Dr. Benj. Morje, Physician to the German Dispensary, N. Y., reports, *N. Y. Med. Record*, July 19, his success with oxalate of cerium in the spasmodic stage of whooping cough. He was induced to try it from the recommendation of Dr. Thos. Clark, in the *Practitioner*, of its value in chronic cough. He reports three of the ten cases in which he has used it successfully—seven males and three females, of ages ranging from one to seven years. The dose was from one-half a grain to three grains according to circumstances, once a day, before breakfast. The practice was to continue the medication a week after the last spasmodic coughing, so as to obviate any relapse.

He concludes from these ten cases, which, though a small number, are yet enough, he thinks, to justify some deductions, that:

1. It decreases the attacks, and thereby reduces the violence of the disease, often checking it instantly.
2. It is easily administered, as only one dose is required in twenty-four hours.
3. Nocturnal quietude is insured.
4. The possibility of complications is lessened.

Salicylic Acid and Salicylate of Soda in Sciatica and Neuralgia.—Dr. S. L. Abbot, *Boston Medical and Surgical Journal*, July 17, gives an account of three cases of sciatica, and three of acute facial neuralgia, in which salicylic acid and salicylate of soda were employed with success. He says, in conclusion: "These cases seem to show that we have in salicylic acid and its compounds as valuable a remedy in acute neuralgia as in acute rheumatism. Perhaps they indicate a closer affinity between these diseases than has been generally suspected. Two of the patients had had attacks of rheumatism. The cases, with the exception of the third case of sciatica, were all severe, and nothing could be more satisfactory than the very prompt and efficient relief which followed the exhibition of the remedy. In cases of a more chronic character, my experience does not justify me in expecting such complete relief by these remedies alone. A limited experience seems to show that such patients do not tolerate them so well, perhaps on account of the debility, which is so marked a feature in these cases."

THE PHYSIOLOGICAL ACTION OF PLATINUM.—Platinum is one of the metals whose action on the living organism we know very little about. Therapeutically, it is at present nowhere; and yet some recent researches, made by Herr Kebler, at Strasburg, show that its properties are, in some respects, peculiar. The salt used in these experiments was the double chloride of platinum and sodium; a solution which, containing 0.050 platinum in one cubic centimetre, was injected, in varying quantities, under the skin of frogs, and into the veins of warm-blooded animals. In frogs, three to fifty milligrammes of the metal first caused increased general reflex irritability, and then the voluntary movements became paralyzed, while spontaneous convulsive twitchings of the limbs, or of certain groups of muscles, occurred, which ceased on division of the medulla oblongata. In rabbits, twenty-five to fifty milligrammes of platinum, and in dogs, forty-five to ninety-five caused profuse diarrhœa when injected into the jugular vein. In dogs, blood generally appeared in the motions, and vomiting was not uncommon. Hyperæmia of the mucous membrane of the stomach and intestine occurred in rabbits; but in dogs all the abdominal viscera were hyperæmic, even the nerve-trunks being involved, while the digestive mucous membrane was softened and infiltrated with blood. The bladder of dogs, poisoned with large doses of platinum, was sometimes ecchymosed. The action of the salt on the circulation mainly showed itself in a continuous fall in the blood-pressure, with scarcely any alteration in the frequency of the pulse. This fall appears to depend entirely on dilatation of the blood-vessels, especially those of the abdomen, owing to paralysis of the terminations of their peripheral nerve-fibres. The excessive stagnation of blood in the abdominal vessels, due to their dilatation, Kebler regards as the cause of the morbid phenomena occurring in the digestive tract. He is also inclined to refer the nervous symptoms, met with in frogs, in part, at least, to the vascular dilatation, though unable to exclude the possibility of a direct action of the platinum on the central nervous system. We record these experiments without suggesting in what way they may be made therapeutically serviceable. One thing, however, seems clear, that the peculiar action of platinum was scarcely likely to prove of much value in the treatment of syphilis, the disease in which it has, as yet, been chiefly tried. (*Med. Times and Gazette*, Feb. 8, 1879.) (*Practitioner*, July.)

BROMIDE OF POTASSIUM.—Dr. A. Hughes Bennett, *British Med. Journal*, June 7, concludes his lectures on the analysis of one hundred cases of epilepsy, all of which were treated with the bromides, with the special purpose of testing the remedy in a series of successive and unselected cases.

All general measures requisite were also resorted to, such as looking after the general health, diet, palliating all unfavorable influences of surrounding, etc., as far as possible. The dose given was, as a rule, in the average adult case, thirty grains *ter die*, and a combination of the bromides of potassium and ammonium, twice as much of the former as of the latter, with aromatic spirits of ammonia and water was found a convenient and efficacious prescription. The first dose was taken on arising in the morning, the second in the middle of the day on an empty stomach, and the third the last thing before retiring. Sometimes, if apparently needed, the dose was gradually increased to sixty or even ninety grains three times a day. After two or three months the quantity was gradually diminished to the smallest quantity found efficacious in warding off the paroxysms, and continued at this amount for many months.

We would say here that the above doses are considerably larger than we have ourselves found needful in the great majority of cases of epilepsy.

Dr. Bennett continues as follows. Of the hundred cases treated in this way, it may be stated in general terms that, with only one or two exceptions, the bromides have had the effect of materially modifying the frequency and severity of the epileptic seizures. At the same time, opportunity was not afforded in all of these to test the efficacy of the treatment for a sufficient length of time. In the following tabular statements, the minimum period during which any patient was under observation and treatment was six months, and this may be considered ample time to demonstrate the therapeutical action of the drugs on the attacks. Therefore, in forty cases under treatment at least six months, and most of them for a longer period, the following tables indicate: 1. The average number of attacks per month prior to treatment, and 2. The average number of attacks during six months of treatment by the bromides.

EPILEPSIA GRAVIOR.

No. of Cases	Average number of attacks per month before treatment.	Average number of attacks per month after treatment.	Number of attacks during six months of treatment.	No. of Cases	Average number of attacks per month before treatment.	Average number of attacks per month after treatment.	Number of attacks during six months of treatment
1	180	60	--	17	4	0	5
2	120	4	--	18	4	2	--
3	60	8	--	19	4	2	--
4	30	0	7	20	2	0	0
5	30	4	--	21	2	0	2
6	16	2	--	22	2	0	4
7	12	1	--	23	2	1	--
8	12	2	--	24	1	0	0
9	8	0	0	25	1	0	0
10	8	0	0	26	1	0	0
11	8	0	4	27	1	0	1
12	8	1	--	28	1	0	2
13	5	0	0	29	1	0	2
14	4	0	0	30	1	0	4
15	4	0	1	31	1	1	--
16	4	0	2	32	1	2	--

EPILEPSIA MITIOR.

33	900	30	--	37	30	6	0
34	300	3	--	38	30	30	0
35	120	30	--	39	20	0	4
36	60	0	0	40	1	0	1

A consideration of these tables demonstrates the very remarkable influence of the bromides on epileptic attacks. In no less than 92.5 per cent., the seizures were either entirely arrested during the period of observation, or greatly modified in frequency and severity. In 22.5 per cent., the paroxysms were completly checked during the whole time of treatment, the minimum length of which was six months, and in most for a longer period. For example, case No. 9 was that of a boy aged 12, who had suffered from epilepsia gravior all his life, having had for some time an average of two attacks per week. He was under treatment for nine months, and during that time he did not have a single seizure. Again, case No. 36 was that of a man aged 30, who had suffered from epilepsia mitior for a year, having two or three attacks regularly every day. During six months of treatment, he had no seizures of any kind.

In 70 per cent., although the attacks were not completely arrested, the reduction in their number was in most cases very marked, as will be seen on consulting the tables. For example, case No. 2 was that of a girl aged 19, who had been subject to attacks of epilepsia gravior since childhood, and who for some time past had had four seizures every day. After treatment, the paroxysms were reduced to one a week. Again, case No. 33 was a girl aged 9, who had suffered from epilepsia mitior for five years. She had about thirty attacks every twenty-four hours. Treatment reduced this to one seizure a day. This modification of the frequency of the attacks took place in every proportion, as will be seen in the tables, in the majority of cases, however, being very remarkable. This is all the more apparent when it is remembered that, in the above general estimate, no account is taken of special adverse circumstances, such as very chronic cases, hereditary transmission, co-existence with disease of the nervous system, and other varying conditions, the accompaniments or causes of the attacks. Another point which may be mentioned is that, so far as the limited number of cases of the *petit mal* permit us to judge, the benefit derived from treatment is nearly equal to that of the *grand mal*, there being only one case out of eight which received no benefit.

This modification of symptoms, if not scientifically curative, is of the greatest possible importance, and nothing can exceed the advantage accruing both to the patient and his friends, by even the relief of the much dreaded fits. Let us take, as an example of this, case 10. This was a young lady, aged 12, who, while at the Cape, was supposed to have had a sunstroke when fourteen months old. Since that time, she had suffered from constant attacks of the severest forms of epilepsia gravior. The minimum frequency of these seizures had been two a week, but for months together she had had several every day.

About every three months, she fell into a sort of *status epilepticus*, in which she had a succession of fits lasting for twenty-four or more hours. It is difficult to describe the distress, anxiety, and constant alarm produced in a private family by one of its members being in this condition. In the present instance, besides general treatment, twelve grains of bromide of potassium were administered three times a day, and the patient has taken this continuously for ten months. During this time she has not had a single seizure except during one week, when, the medicine having been discontinued, she had three attacks. The general health has greatly improved, the patient has grown stout and tall, and menstruation has occurred for the first time, and altogether she is more healthy and robust than she has been for years. This child is not cured, but it is unnecessary to point out the benefit derived both by her friends and herself from even the modification of the paroxysms.

Of the forty cases which compose the tables, in only three were there no beneficial changes on the attacks as a result of treatment. There was nothing special in any of these to indicate why the remedies should have failed, as the following notes may show:

Case No. 31 was a woman aged 25; suffering from epilepsy for six years. Attacks of epilepsia gravior came on about once a month; of epilepsia mitior, about ten or twelve times a day. She was a very delicate and nervous person; otherwise there was nothing special. Her family was healthy.

Case No. 32 was a man aged 32. He had had epilepsy for ten years; attacks of epilepsia gravior once a month—otherwise, nothing special. His family was healthy.

Case No. 38, a man aged 37. He had had epilepsy for ten years. Attacks of epilepsia mitior came on once a day. He was a delicate, weak person. His family was healthy.

Not only do the bromides materially modify the frequency of epileptic attacks, but they often diminish the severity of those which occur. They also improve, in many respects, the general health; and persons who suffered from headache, nervousness, and other ailments, are often greatly relieved in these respects.

The administration of the drugs may arrest the seizures for many months, and the moment they are discontinued, the attacks at once return, indicating that it is these agents which keep the paroxysms in abeyance, and that their action is not permanent.

What effects has a prolonged use of the bromides on the general health? Of the forty cases, which for a period of at least six months were continuously under the influence of these drugs, the following gives a general idea of the results:

General health not affected	25
Symptoms of bromism	15
Enfeebled or weakened body	10
Depressed or weakened mind	8
Eruption	6
Died	1

It will thus be seen that, in 62.5 per cent. of cases, the prolonged use of the bromides, sufficient to ward off or greatly modify the epileptic attacks, did not produce any physiological effects, or in any way influence the general health. In 35 per cent. some symptoms of bromism were produced, namely: in 25 per cent. there were weakness and languor of body, loss of appetite, and the usual physical symptoms; in 20 per cent. there were depression of the mental faculties into dullness, apathy, tendency to sleep, and so on; and in 15 per cent. there were well marked signs of the bromide rash. One patient died while taking large doses, but whether as a result of the remedy or of the disease, it is very difficult to determine. As a rule, however, the symptoms of bromism were slight, and their effects temporary, and rapidly disappeared on discontinuing the drug for a time.

It may be said in conclusion that, in the bromide of potassium, we possess a valuable agent for suppressing the most dangerous symptoms of one of the most terrible maladies to which human flesh is heir; and further experience may enable us, through its influence, to effect a complete cure of the disease itself.

There are other methods of treatment of epilepsy, both by medicinal agents and by derivatives, such as the seton and actual cautery, which I am disposed to believe in some instances of the greatest importance, a consideration of which, space compels me, at present, to postpone.

CURARE IN HYDROPHOBIA.—There have been several reported cures of hydrophobia within the past two or three years; one of the latest is reported by Dr. Ad. Offenberg, Wickrath (Rhein-Preussen), and translated by Dr. S. W. Williams, *N. Y. Med. Record*, Aug. 9:

A servant girl was bitten, on the 28th of July, 1874, in the heel by a Spitz dog, pronounced mad by a veterinary surgeon. Other cases of rabies had occurred in the neighborhood, and the man in whose house she was working died hydrophobic from the effects of a dog-bite only two days after the girl herself was bitten. The wound was treated locally and kept open, and she took remedies popular in the region to prevent hydrophobia. On the sixteenth of October she first gave signs of hydrophobic spasms, which in the course of the night became very pronounced. Injection of .02 (= ⅓ grain) morphia and chloroform inhalation being ineffectual, and the diagnosis of hydrophobia being considered established, Dr. Offenberg began to use curare hypodermically, a solution of the strength of 0.5 : 10 of distilled water (= grs. 7½ : ʒ iiss). The effectiveness of this solution had been previously tested on frogs, and also in two cases of traumatic tetanus.

At first .02 (= ⅓ gr.) was injected without effect, and the dose was repeated in fifteen minutes, with the result of diminishing the general muscular tremor and reducing the pulse between the convulsions, and also of increasing the intervals of these latter. At midnight nearly half a grain ($\frac{6}{13}$) of curare was given, followed by two other similar doses at intervals of an hour, then another in half an hour, at 2.30 a.m. Oct. 17, and in fifty minutes more (3.20 a.m.) still another. The result of all this was considerable improvement in the patient's spirits and general comfort between the spasms, the diminution of their severity and the lengthening of the intervals between them, and, after the last dose, almost complete muscular paralysis, not, however, involving respiration, and cessation of the spasms. During the next day the paralysis disappeared almost entirely, but slight convulsive phenomena reappeared, excited by drinking, swallowing, etc., and spasmodic muscular twitchings, but not serious. The next day or two there occurred temporary paralysis and some occasional slight convulsions, and numerous head symptoms persisted for several days. But from the 25th of October the recovery was gradual and steady. She was discharged from the hospital on the 4th of December, 1874, in good general health, but not strong, and with weakness of the eyes, which had troubled her during the attack. In January, 1875, she resumed her work, though not yet strong, and recovery was still slow. Two years later she was in perfect health.

The dubious point in this case, as in all the cures of this disease that we have seen reported, is the question whether this was an actual or a hysterical case. The fact is stated in the account that the patient was much alarmed as to her condition before she was taken ill, and that her uneasiness persisted even after her recovery. The case has not the conclusive character that it would have were all such moral complications excluded in the history. Still we are inclined to believe that hydrophobia is in all cases very much a mental disease, *i. e.*, the mind is affected, even in the lower animals. There is much to encourage us, therefore, in these later clinical experiences with this fearful disorder.

IN VINO VERITAS.—In the *Vierteljahrschrift für gerichtl. Medicin*, July, Dr. Westphal reports the case of a murderer who was alleged to be partially insane. Among the tests was the administration, on two occasions, of sufficient brandy to make him quite drunk. In this condition of intoxication, he plied him with a number of questions, believing that if his delusions were simulated, he could not keep up the deceit when so much in liquor. As the man still persisted in his insane statements, the expert concluded that he was really demented. (*Med. and Surg. Reporter*, Aug. 9.)

BROMOHYDRATE OF CONIINE.—At the sessions of the Acad. des Sciences, Paris, July 14, 21 and 28 (rep. in *Bull. gen. de Thérap.*, Aug. 15), M. J. L. Prevost read a communication, of which the following are the conclusions drawn from experiments on various species of animals:

1. The paralysis produced by bromohydrate of quinine is the result of the paralysis of the motor nerves, which also lose their excitability.

2. When we interrupt the circulation in the posterior portion of the body of a frog, sparing the nerves, and then introduce, hypodermically, in the back, a dose of from 0.015 to 0.02 of bromohydrate of coniine, the nerves of the posterior members remain excitable, and these posterior limbs react to excitations made on the anterior members above the ligature, they being also paralyzed by the poison.

3. This experiment is made more striking by strychnizing the animal; in that case we can observe, simultaneously, the effects of the two poisons on the same animal.

4. The pneumogastric is paralyzed before the other nerves, and its excitability reappears more promptly than that of the other nerves, during the period of the elimination of the poison.

5. The urinary, salivary and lachrymal secretions are excited by bromohydrate of coniine.

6. I have been able to experimentally determine the passage of the drug in the urine. The urine of a cat, poisoned with bromohydrate of coniine, evaporated in a warm bath to a syrupy consistence, and injected under the

skin in several frogs, produced in these animals the characteristic symptoms of poisoning by bromohydrate of coniine.

7. The glandular nerves still preserve their excitability, and cause secretion when electrized. After the vagi and the nerves of the striped muscles have lost their excitability, the electric excitation of the cervical sympathetic and the tympanico-lingual, causes a flow of saliva. The excitation of the peripheral end of the nerves of the fore-limb produced secretion of sweat in the palmar surface of a cat's paw, after it had failed to arouse muscular contractions.

8. In cold-blooded animals, poisoned by bromohydrate of quinine, and subjected to artificial respiration, the heart showed great resisting power, dying last of all; it continued to pulsate longer than a normal heart, after cessation of artificial respiration, or when separated from the body.

In rabbits and cats, the direct electrization of the heart by a strong induction current, did not produce its paralysis when poisoning was carried to the point of complete loss of the excitability of the sciatic nerve. This experiment failed in a cock. When in rabbits the excitability of the sciatic was not completely destroyed, electrization of the heart would produce paralysis; but repeated attempts were needed to produce this effect.

9. It is very doubtful whether the nerve-centres are directly affected by bromohydrate of coniine. The convulsions observed in warm-blooded animals in the later period of the poisoning, are the result of the asphyxia due to the paralysis of the mechanical factors of respiration. These convulsions may be avoided by means of artificial respiration.

10. Muscular contractility is not at all affected by bromohydrate of coniine.

THE PNEUMOGASTRIC AND THE ACTION OF DIGITALINE.—At the session of the Acad. des Sciences, Paris, June 2d, M. Ch. Robin presented a communication by M. Cadiat, on the influence of the pneumogastric, and the action of digitaline on the movements of the heart in the squaloids, which is thus reported in *L'Union Médicale:*

"Certain of the squalidæ, among others the roussette (*Scyllium caniculum, H. D.*), preserve an excessive nervous excitability long after death, together with simultaneous violent cardiac pulsations, which last ten or twelve hours after cessation of all spontaneous movement. This animal seems, therefore, to offer very favorable conditions for studying the action of the pneumogastric and the effects of digitaline. The results I have obtained are presented in evidence.

1. "The pulsations of the heart, which in the conditions of artificial respiration in which I had placed the sharks for these experiments, rise to ten or twelve a minute, increase rapidly after the section of one or both pneumogastrics, or the destruction of the medulla, to twenty-eight or thirty in the same space of time.

2. "If we cut the pneumogastric between the cranium and the organ of the branchial nerves and then excite the peripheral portion, we obtain an immediate arrest in diastole, whatever be the method of excitation. The excitation of the central end has no effect on the cardiac movements.

"This experiment may be repeated for hours on the same animal, either before or after death, that is, after disappearance of all voluntary movements.

3. "The pneumogastric giving out, after it leaves the cranium, as many branches as there are branchial rays, the action of arrest of the heart is so much the more energetic as we apply the excitation to a part including the largest number of these branches. With only one branchial filament we can hardly arrest the heart. The nerve of the third branchial pair seems to have the most effect.

4. "When the excitability of the pneumogastric is exhausted, its excitation does not stop the heart."

Action of Digitaline.—The experiments with digitaline confirm incontestably the results of M. Bröhm and M. Vulpian.

In opposition to Traube, who thought that digitalis acted on the heart through the nervous system, M. Vulpian recognized the fact that digitalis, like the *Upas antiar* was a direct cardiac poison; but many objections may be raised to the experiments he reports in support of this view. In one of them in fact, the heart was arrested in systole under the influence of digitaline, but it is not said that its nervous excitability still persisted at the time. In another, of two animals, the one not yet poisoned succumbed with cardiac arrest from the mere effect of the operation, before absorbing any digitaline. These experiments upon sharks are much more conclusive on account of the great vitality of the animals.

1. "I opened the pericardium of a roussette (*Scyllium caniculum*) and cut the pneumogastric. The heart beat 25 per minute; some drops of solution of digitaline were then dropped on the heart. The movements of the organ were at first quickened, then the diastoles gradually diminished; the heart appeared to sink down into the pericardium. Suddenly it stopped in systole in a sort of tetanic condition, and electrical excitation could arouse no pulsation.

"But the animal had not yet died; the nerves and muscles were still excitable; it struggled violently when lifted and put into a basin, the heart being absolutely quiet; it continued its natatory movements for over half an hour.

2. "I repeated this experiment on another of these sharks, but instead of cutting the pneumogastric, I destroyed the medulla. This mutilation also accelerated the cardiac movements, which became as frequent as when the pneumogastric was cut. A few drops of digitaline are poured on the pericardium. The heart continued to beat; then suddenly it ceased in systole.

"The spontaneous movements still continued.

3. "I cut the left pneumogastric one day, and the next day cut the right also. The excitation of one of these nerves did not arrest the heart any more, a fact due, doubtless, to the accelerating influence of the divided symmetrical nerve.

"A large dose of digitaline was then injected into the peritoneum; then, a quarter of an hour later, I opened the pericardium. The heart-pulse was twenty-six to thirty-one per minute, but the diastoles gradually diminished and the heart suddenly stopped in systole.

"The nervous system was still excitable, as the animal swam when placed in water.

4. "In another of these sharks, I commenced with a strong injection of digitaline. Ten minutes later I laid bare the pneumogastric and the heart. The latter was absolutely immobile in systole; but at this moment the excitation caused progressively a dilatation and excessive repletion of the organ. As we might attribute this to the influence of muscular contractions, I passed a current along the cord, causing violent motions but no cardiac dilatation.

"This experiment shows that the paralysant action of the pneumogastric is sufficient to still arouse a tetanized heart.

"The heart having been entirely quiet and in systole for half an hour, we put the animal in water and it at once traversed a space of two or three metres.

"*Conclusion:* Digitaline administered to animals in toxic doses, acts as a cardiac poison. It acts directly on this organ, causing, as many authors have already stated, a tetanization of the ventricle and diastole of the auricle. It has no action on either the nerve centres, the peripheral nerves or the muscles."

NUX VOMICA AS A REMEDY FOR VERTIGO.—Dr. Mader, *Wiener Med. Blätter*, 8-13, II. Jahrg. (*Deutsch Med. Wochenschr.*, July 19.)

The author has employed extract of nux vomica (0.1—0.2 per diem, divided into two doses,) in a considerable number of cases for the relief of vertigo. Its remarkable effects are shown in twenty-six clinical histories. For theoretical explanation these must be divided into two classes. On the one hand we have a morbid affection in the sensory sphere with secondary motor disorders, and then these latter may occur primarily as the motor association centres are morbidly irritated and produce slight unconscious active movements, of which we are only passively conscious. That nux vomica, as it appears, acts directly in those cases in which the motor sphere is involved, is easily understood from its known physiological peculiarities. If one now understands that the cause of the irritation of the motor region of the nervous system is usually from a local disorder of the circulation, for instance, a hyperæmia, then the action of the preparation in question would indicate that it contracts the vessels. The many weak points for attack this explanation may also afford, as the author himself admits, should have the result to excite further experiments with the agent.

HYPODERMIC INJECTION OF FOWLER'S SOLUTION IN CHOREA OF CHILDREN.—M. Henri Garin gives in a thesis the result of the treatment practiced by Doctor L. Perroud, of Lyons.

The method practiced was to inject four or five drops of Fowler's solution, as a rule, every two or three days, sometimes, however, oftener.

The reasons which lead the author to prefer this method of treating the disease are:

I. Because the gastric disturbances, so troublesome when the medicine is given by the stomach, are avoided.

II. Because the good effects of the drug are generally obtained more rapidly.

III. Because it is only necessary to employ small doses, administered every two or three days.

The irritation at the point of injection is trifling. Sometimes on account of idiosyncrasy, the arsenic is not tolerated, and symptoms of arsenical poisoning are observed. But these cases are rare, especially in children, who, as a rule, bear arsenic very well. Under the influence of this treatment, rapid improvement is the rule in all cases.

This amelioration of the symptoms cannot be ascribed to acupuncture, as the injection of water does not have the same result.

As the chorea becomes better the patient gains flesh, and the solid matter excreted by the kidneys diminished in quantity.

By the use of arsenical injections 16 patients were cured, after an average of 32 days of treatment, and 18 hypodermic injections.

These 10 cases were put on a purely arsenical treatment, and they were all simple and recent cases. In 13 other cases submitted to the treatment, but which had been under other treatment, 10 were cured. The time necessary to effect a cure was more than in the cases first mentioned. These 13 were, as a rule, old cases and relapses.

From the preceding we can conclude that arsenic is more efficacious in simple and recent cases than in old and inveterate ones, which is contrary to the assertion of Aran, Ziemssen, etc.

The number of cases observed was 33, all girls, ranging in age from 4½ to 14½ years.—*Thése de Lyon*, No. 14.—*Lancet and Clinic.*

THE ACTION OF BRUCINE AND STRYCHNINE ON THE MOTOR NERVES.—Dr. Lautenbach, in the *Medical Times* of August 2d, reaches the following conclusions on the above subject, viz.:

Brucine treated with acetic acid and injected into a Rana temporaria, in the proportion of one part of the alkaloid to about fifteen hundred parts by weight of the frog, produces a non-excitability of the motor nerves through a direct action on these structures.

Brucine treated with hydrochloric acid produces the same effect in the Rana temporaria; but in the Rana esculenta it, in much smaller doses, paralyzes the motor nerves without having previously induced tetanus.

Strychnine treated with acetic or hydrochloric acid produces in the Rana temporaria, paralysis of the motor nerves in one-sixth the dose necessary for brucine to produce the same effect.

The following are the titles of a few recently published articles on the Therapeutics of the Nervous System and Mind:

KENYON, Treatment of Eclampsia with Veratrum Viride, *N. Y. Med. Jour.*, Oct.—BUSEY, Anæsthesia and Hot Water in the Treatment of Torticollis, *N. Y. Med. Rec.*, Sept. 27.—SAVAGE, Hyoscyamine and its Uses, *Jour. of Ment. Science*, July.—SCHAEFFER, Asthma and its Treatment, *Deutsch. Med. Wochenschr.*, No. 32.—STONE, The Physiological Action of Carbolic Acid on the Nervous System, *Phil. Med. Times*, Sept. 27.—MENDEL, The Therapeutic Use of Hyoscyamine in Psychoses, *Allg. Zeitschr. f. Psych.*, XXXVI., II. & III.

BOOKS, ETC., RECEIVED.

Lehrbuch der Psychiatrie auf Klinischer Grundlage, für Practische Aerzte und Studirende. Von Dr. R. v. Krafft-Ebing. Drei Bände. Band I: Die Allgemeine Pathologie und Therapie des Irrseins. 276 pages. Band II: Die Specielle Pathologie und Therapie des Irrseins. 214 pages. Stuttgart, 1879.

Anatomische Studien an Verbrecher-Gehirnen. Für Anthropologen, Mediciner, Juristen und Psychologen, bearbeitet von Moriz Benedikt. Mit 12 Tafeln und 8 Holzschnitten. Wien, 1879. 149 pages.

Handbuch der Physiologie. Herausgegeben von Dr. L. Hermann. Zweiter Band: Physiologie des Nervensystems. Erster Theil: Allgemeine Nerven-Physiologie. Von L. Hermann. Zweiter Theil: Specielle Nerven-Physiologie. Von Sigm. Mayer. Leipsig, 1879. 288 pages.

Dictionnaire Encyclopédique des Sciences Médicales. Dir. A. Dechambre. Premiere serie, A–E. Tome Vingt-troisieme, Premiere Partie, Créches–Cristallin. Paris, 1879. 352 pages.

Nouveau Dictionnaire de Médicine et de Chirurgie Pratiques. Dir. Dr. Jaccond. Tome Vingt-septieme, Pess–Pie. Paris, 1879.

Memoire sur les Affections Syphilitiques Precoćes des Centres Nerveux. Par Ch. Mauriac. Paris, 1879. 199 pages.

The Brain and its Diseases. Syphilis of the Brain and Spinal Cord, showing the part which this agent plays in the production of Paralysis, Epilepsy, Insanity, Headache, Neuralgia, Hysteria, Hypochondriasis, and other Mental and Nervous Derangements. By Thomas Stretch Dowse, M. D. New York, G. P. Putnam's Sons, 1879. 144 pages. Chicago, Jansen, McClurg & Co.

Morphologie du Cerveau. Pour l'Etude des Localizations des centres excito-moteurs des Hemispheres et de l'Operation du Trepan. Par le Docteur E. Gavoy. (Avic Planches.) Alger., 1879. (Extr. du *Jour. de Medicine et Pharmacie d'Algerie.*)

Sulla Sintomatologia e sulla Diagnosi delle Lesioni Corticali della Zona Motrice. Pel Dottor Dario Maragliano. Reggio-Emilia. 1878.

Perizia sulla Stato di Mente di G. Passanante (Tentativo di Regicidio). Dei Professori Tommasi, Verga, Biffi, Buonoma, Tamburini (Relatore, Tamburini). Reggio-Emilia. 1879.

Della Sifilide Epilettifome. Pel Dott. Celsa Pelizzari, Assistente alla clinica Sifilopatica nell' Istituto Superiore di Firenze. Firenze, 1879.

Des Alienes Voleurs. Non-existence de la Kleptomanie et des Monomanies en General commes entites Morbides. Thése pour le Doctorat en Médecine, presentée et soutenue le 23 Juin, 1879. Par Th. Govroy. Paris, 1879.

Real-Encyclopädie der Gesammten Heilkunde. Medicinisch-chirurgisches Handwörterbuch für praktische Aerzte. Herausgegeben von Dr. Albert Eulenburg, ord. Professor an der Universität Greifswald. Mit Zahlreichen Illustrationen in Holzschnitt. I. Lieferung, Band I., Bogen 1–5. Wien, 1880.

Transactions of the Pathological Society of Philadelphia. Volume eighth, containing the Reports of the Proceedings from Sept., 1877 to July, 1878. Edited by J. Henry C. Simes, M. D., Philadelphia. Printed for the Society by J. B. Lippincott & Co., 1879. Chicago, Jansen, McClurg & Co.

Reports to the St. Louis Medical Society on Yellow Fever, consisting of the Report of the Committee appointed to inquire into the relations of the Epidemic of 1878 to the city of St. Louis. By W. Hutson Ford, A. M., M. D. Revised by the Committee on Publication. St. Louis: Geo. O. Rumbold & Co., 1879.

Transactions of the Medical Association of Georgia, Thirtieth Annual Session, Rome, April 16, 17, 18, 1879. Jas. P. Harrison & Co., Printers and Binders, Atlanta, Georgia, 1879.

Transactions of the Medical Society of the State of Tennessee at its Forty-Sixth Annual Meeting, 1879. Nashville, Tennessee. Printed at "The American" Book and Job Rooms, 1879.

Twenty-Second Annual Report of the Board of Directors for Public Institutions of the city of Boston, for the financial years 1878–79. Boston House of Reformation Printing Department, Deer Island, Boston Harbor, 1879.

The Future Influence of the John Hopkins Hospital on the Medical Profession of Baltimore. By John Van Bibber, M. D. Baltimore, Jones & Company, Printers, 1879.

A case of Anterior Spinal Paralysis with formation of Vacuoles in the Ganglion Cells of the Spinal Cord. Read before the American Neurological Association, New York, June 19, 1879. By Robert T. Edes, M. D. (Reprinted from the Boston Medical and Surgical Journal, July 24, 1879.) Cambridge: Printed at the Riverside Press, 1879.

Some points connected with the Question of Responsibility as it Relates to the Partially Insane. By T. L. Wright, M. D. Bellefontaine, Ohio.

Reflex Cerebral Hyperæmia. By C. H. Hughes, M. D. Read before the St. Louis Medical Society. (Reprinted from the St. Louis Medical and Surgical Journal, June, 1879.)

Annual Address on the Relation of Neurasthenia to Diseases of the Womb. By William Goodell, M. A., M. D., Philadelphia, Pa. (Reprint from Volume III., Gynecological Transactions, 1879.)

Views suggested by the Study of the Etiology of Puerperal Eclampsia. By J. K. Bauduy, M. D., St. Louis. Read before the Obstetrical and Gynecological Society of St. Louis, May 15, 1879. (Reprinted from the St. Louis Courier of Medicine, August, 1879.)

The Construction, Organization and Equipment of Hospitals for the Insane. By William A. Hammond, M. D. Read by invitation before the Connecticut Medical Society at Hartford, May 29, 1879. New York, G. P. Putnam's Sons, 1879.

The Tendon-Reflex in the Insane. By J. C. Shaw, M. D. (Reprinted from Archives of Medicine, August, 1879.) New York, G. P. Putnam's Sons, 1879.

Buildings for Insane Criminals. By Walter Channing, M.D., Boston. Read at the Conference of Charities, in Chicago, June 11, 1879. (Reprinted from the Proceedings.) Boston: Rand, Avery & Co., 1879.

Emotional Insanity. By J. K. Bauduy, M. D. (Reprinted from the St. Louis Medical and Surgical Journal, April, 1879.) St. Louis: Geo. O. Rumbold & Co., 1879.

Report of the Board of Trustees of the Michigan Asylum for the Insane, for the years 1877–78. Lansing, W. S. George & Co., State Printers, 1879.

Phenol, its Poisonous Effects, and the Soluble Sulphates as Antidotes. By David Cerna, M. D., Ph. D. (Reprinted from The Philadelphia Medical Times.)

Dermatitis Venenata; or, Rhus Toxicodendron and its action. By Roswell Park A. M., M. D. (Reprinted from Archives of Dermatology, July, 1879.)

The Hot Springs of Arkansas as a Health Resort; their Waters as Remedial Agents. By J. M. Keller, M. D. (Reprinted from the St. Louis Medical and Surgical Journal, August, 1879.) St. Louis: Geo. O. Rumbold & Co., 1879.

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Archiv fuer Anatomie, Physiologie, und Wissenschaftl. Medicin.
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Archiv fuer die Gesammte Physiologie der Menschen und Thiere.
Archiv f. Psychiatrie u. Nervenkrankheiten.
Brain.
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Cronica Med. Quirurg. de la Habana.
Dublin Journal of Medicine and Surgery.
Deutsche Medicinische Wochenschrift.
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Philadelphia Medical Times.
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